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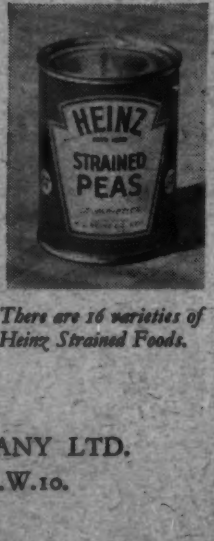

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BRITISH MEDICAL JOURNAL

LONDON SATURDAY MAY 17 1952

VALVOTOMY FOR MITRAL STENOSIS

A FURTHER REPORT, ON 100 CASES

BY

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Our interest in valvotomy for mitral stenosis extends back over several years, although it was not until September, 1948, that our first patient was operated on. In June, 1950, we were able to report our experiences in nine patients, seven with successful operations (Baker, Brock, and Campbell, 1950). We are now able to report the immediate results on 100 patients treated by operation, with a more detailed analysis and follow-up of the first 50, the last of whom was operated upon in May, 1951, so that all have been followed for six months, the majority for more than a year, and some for two to three years. Apart from two patients at the Johns Hopkins Hospital, Baltimore, the operations were done at either Guy's Hospital or the Brompton Hospital, and all by one surgeon. For the purpose of analysis and follow-up the two Baltimore cases have been omitted from the first 50.

In previous papers we have followed common usage in writing of pulmonary or mitral "valvulotomy," but it is a valve and not a valvule that is divided, and we think the term "valvotomy" is more correct, even though it is still a hybrid formation. The operation really does more than splitting the commissures, for if the valve cusps are reasonably supple the operation may enable them to close better and more normally, but we have thought it better to reserve the term "valvoplasty" for more elaborate operations that may be possible in the future.

Present Position of Mitral Valvotomy

Our earlier paper contained a fairly full presentation of the problem of mitral valvotomy up to that time, and there is no need for repetition here. The great improvement observed in seven of the nine patients enabled us to say with some confidence that the operative relief of mitral stenosis was a practical procedure with a reasonably low mortality, and that it could justifiably be recommended in suitable cases and would probably have a wider application. Our subsequent experience reported here with 100 cases and with 32 good or excellent results in the follow-up of the first 50, as well as the experience of several other clinics, amply confirms these views.

The relatively low mortality of the operation deserves immediate emphasis. Thus of our first 20 patients, 7 died; only one more patient died in the next 30, making 8 deaths in the first 50 cases, and only 5 more in the next 50, making 13 deaths in the first 100 cases. Even severely ill patients with advanced mitral stenosis will stand cardiotomy well, *provided the mechanical obstruction within the heart is relieved by the operation.*

As soon as our first report appeared the great need for operative relief of mitral stenosis was borne vividly upon us by the large number of patients presenting themselves for consideration, the requests coming from cardiologists, from general practitioners, and from the patients themselves. A future that previously seemed bleak for many patients, generally under 40 and often under 30 years of age, has been completely changed. It is only in dealing with a large number of these patients that one realizes the true extent of the tragedies that mitral stenosis can produce.

Successful surgical treatment of mitral stenosis has disclosed a problem of great extent. Congenital heart lesions, for instance, constitute only 2% of all cases of heart disease, and it is fair to say that their operative relief is best undertaken in a few special centres. By contrast, rheumatism causes 25% of cases of heart disease in hospital (Wood, 1950), and in half of them mitral stenosis is the predominant lesion. It is not possible for all the patients for whom operation is required to be dealt with in a few special centres. Mitral valvotomy is now a standard procedure that should be practised in all thoracic surgical clinics in large centres.

Although a new field of surgery has been opened up, the problem is even larger than the purely surgical conception. In some respects it is comparable with that of pulmonary tuberculosis, in which surgery has come to play a large and increasing part, but is still only one factor in the general management. Many patients with mitral stenosis, as many with pulmonary tuberculosis, will never need operation, but must have medical treatment. Close co-operation between surgeon and physician is essential, and when operation is needed the selection of the best time for surgery comes from

discussion between the surgeon and the physician who has guided the patient up to operation and will continue to advise him afterwards.

In no case in this series has the decision to operate been made solely by the surgeon. Even when the patients have been referred direct to the surgeon (as now happens not infrequently and somewhat significantly) they have always been transferred to the care of one or other of the cardiologists for a full and independent assessment.

Grading

In assessing the severity of the disease we have graded the patients into five groups, based largely on the capacity for exertion without dyspnoea, but partly also on the general disablement.

When there is no disability the patient is classed in grade 0. Grade 1 means undue breathlessness on hurrying, walking uphill or upstairs, running for a bus, or on other relatively strenuous activity. Patients in this grade lead a virtually normal life, but cannot keep up with their fellows during physical pursuits.

Grade 2 means breathlessness even on moderate exertion. Patients in this grade cannot hurry, although they may be able to walk some miles on the flat at their own speed. Hills and stairs are only managed slowly, and running provokes acute discomfort.

Grade 3 means serious incapacity that interferes with the daily round. The men are unable to do more than light or sedentary work, and the women do their shopping and housework only with difficulty; hills are out of the question, and stairs are avoided or taken in spells. Patients in this grade are usually orthopnoeic, and may have bouts of paroxysmal cardiac dyspnoea or even pulmonary oedema.

Grade 4 is reserved for patients who are completely incapacitated. Housework and shopping are abandoned, the least physical exertion causing great fatigue and breathlessness. Patients in this grade are usually in congestive heart failure or on the verge of pulmonary oedema.

The chief difficulty in grading a patient is caused by the changes that take place from time to time. For example, a woman in grade 0 may develop paroxysmal cardiac dyspnoea during pregnancy and for some months may be in grade 3. Transient upper respiratory tract infection or paroxysmal auricular fibrillation may quickly induce total incapacity (grade 4) in a person previously in grade 1 or 2. Again, grade 4 may be quickly changed to grade 3 as a result of adequate medical treatment. We have tried to grade patients according to their usual condition when they are living at home; not when under transient adverse circumstances, nor after a beneficial period of prolonged medical treatment in hospital.

By using these grades we have come individually to a reasonable agreement in classifying the patients. The assessment at the time of operation in the first 50 patients was made by at least two of us after full investigations, including cardiac catheterization, and was as follows: grade 1, 0; grade 2, 1; grade 3, 19; grade 4, 30.

It will be seen, therefore, that the patients selected for operation up to the present have reached an advanced stage of mitral stenosis.

Results

Deaths

The results in the first 50 cases are summarized in Table I. There were eight deaths. Cerebral embolism occurred in three fatal cases, all with auricular fibrillation (of five years', six years', and three months' duration). The first (G2)* might have survived but for intrathoracic bleeding from heparin therapy. The second (G11), who still had signs from a left hemiplegia three years before, died of an embolism in the right middle cerebral artery. The third (G17) died the day after operation from an embolism of the basilar artery.

TABLE I.—*Results of 100 Mitral Valvotomy Operations*

No. of patients operated on	100
Deaths	13
Follow-up of First 50 Cases	
Deaths	8
Valvotomy not possible	1
Poor result	5
Fair result	4
Good result	15
Excellent result	17

The other five patients, three of them with auricular fibrillation, died within a few hours to three days after operation from circulatory failure. In one (G12) the lateral papillary muscle controlling the anterior cusp of the mitral valve was torn and severe regurgitation resulted. One (G3), a severe risk, lived 36 hours with a gradually falling blood pressure. The other three (G7, G14, G15) did not regain consciousness after the operation and died after a profound fall of blood pressure which failed to return to a normal level: the importance of maintaining the blood pressure must therefore be emphasized. All the eight deaths occurred in the first 22 cases.

In one patient (B2) valvotomy was not carried out because the auricular appendage was solid with firm clot which also extended over a wide area on the adjacent wall of the atrium. It was thought that disturbance of this would probably lead to a fatal massive embolism, as had happened in an earlier case with widespread mural thrombosis (G2).

Poor and Fair Results

In 5 of the 41 patients on whom valvotomy was done there was no improvement or the patient was worse. Two (B12 and G18) developed fibrillation and congestive failure after operation: the first improved with digitalis and returned to light work, but has since relapsed; the second did not respond to digitalis and committed suicide a few months later. These two were men aged 35 and 33 with uncomplicated stenosis, a cardiothoracic ratio (C.T.R.) of 50, and pulmonary congestion; both were expected to do well; both had adequate openings made at operation and no regurgitation was noted. The first had had cardiac pain, which suggests a possible factor; the second was thought to have developed active carditis, though this was not proved. A third patient (G16), with severe pulmonary hypertension, has shown little if any clinical improvement, and catheterization six months after operation showed no fall in pressure (see Table IV) or rise in cardiac output: it is possible that the operation was not successful, for there was much calcification, or that his pulmonary hypertension had become irreversible.

The fourth (B15), a man aged 37, with a history of embolic cerebral disturbance six months before, fibrillating for two months, and with a small heart (C.T.R. 48), had mitral regurgitation, confirmed at operation; this appeared to be successful, but he remained drowsy and developed jargon speech for some time after the operation. He is not improved, and too prolonged fall of blood pressure or cerebral embolism may be partly responsible. The fifth (G33), a woman aged 32, was completely disabled by dyspnoea, repeated haemoptyses, and acute pulmonary attacks. As well as stenosis, she had signs of mitral incompetence, which was confirmed at operation, where the valve orifice was found to be contracted to 1 cm. by 0.5 cm. and surrounded by a hard mass of calcium. The medial commissure alone could be divided, and the opening was increased only to 2 cm.; the regurgitant stream seemed less. Her condition was unaltered by the operation, and the signs of mitral incompetence are more obvious.

Of the remaining 36 patients, we have judged four (B4, G25, G26, G33) as only moderately improved, though they

*Where a patient is referred to in the text the letter refers to Guy's or Brompton, and the figure to the serial number at one or other hospital—for example, G2 means the second operation at Guy's Hospital.

themselves are pleased with the result and have been relieved of orthopnoea and pulmonary episodes. All were incapacitated before operation; now two lead a quiet life at home and two are fit to do clerical work. In two mitral calcification was found: in one it was so extensive and the valve so fibrous that a good result was not expected; in the other it was associated with mitral regurgitation, which was not apparently changed by operation.

Successes

Of the remaining 32 patients we have judged the result as good in 15, where disablement has decreased by two grades, and excellent in 17, where it has decreased by three grades. Some of the results classed as good may later prove to be excellent, for there has not yet been time to test the full improvement. Only twice have we had to revise an early estimate in the opposite direction.

Some concrete examples of results classed as good may make this clearer. A hospital matron, able to hold her post only because her executive work enabled her to sit down, returned to a full day's work, much of it on her feet, without symptoms. A labouring man who had been unable to work because of periods of congestive failure for over a year has since returned to five months of manual work without a day off. A married woman has made a very strenuous tour of France and Italy, doing all she wanted without symptoms, but her result is classed as good only because her disability was graded as 2 before operation; she had, however, been dyspnoeic for nine years with attacks of cardiac asthma and haemoptysis for 10 months.

The 17 patients in whom the results have been adjudged excellent have all improved three grades of disability. This means that those with complete disablement have returned to a full life, either in employment or at housework. Some have protested that they have no symptoms whatever, and need reminding that they must not consider themselves as perfectly normal people. They have passed through a severe winter without being upset by respiratory infections; they have walked up to five miles (8 km.); and a rapid gain in weight of two stone (12.7 kg.) is not an isolated instance. Some previously prohibited by their symptoms from sexual activity have resumed a normal married life. The physical improvement often alters the whole outlook—from resignation or despair to hope and a full savour of life. The women are delighted to return to dancing, and the men to cultivate their gardens. Improvement is immediate and they lose their orthopnoea and "tight breathing" from the second day; one patient who could not walk the length of the ward before operation, when convalescing ran 30 yards (27 metres) for his last bus, missed it and walked the necessary four miles (6.4 km.) without symptoms. The functional improvement in some of these patients has been checked by a second catheterization after operation, and this has shown a large fall in the pulmonary artery pressure (see Table IV).

The demonstration that mitral valvotomy can be performed successfully on very ill patients with a low mortality and with a good prospect of success is most important. Once this was established it was possible, with increasing confidence, to advise the operation more widely and in a greater variety of cases. An operation for mitral stenosis has for a quarter of a century been looked on as a desperate and dangerous venture. It is remarkable that now the pass has been carried the operation is seen to be one of relative safety. No longer is it necessary to reserve operation for those advanced cases that are soon going to prove fatal without it: such cases clearly deserve help, and in our series have not been denied it. To-day there is no need to delay operation on earlier cases through undue fear of the operative risk, and as our experience has extended we have been encouraged to perform valvotomy earlier in the disease. Our recent low mortality is not, however, due to early cases being operated upon, for the number of severe and even desperate cases has so largely occupied our operating resources that it has not been possible so far to include more than a few of the earlier less severe cases.

Analysis of Results in Relation to Selection of Patients

It would be more logical to discuss the results and the selection of patients separately, but to avoid much repetition they are taken together. Greater and more varied experience will be needed before precise statements can be made about the correct selection of patients, but much can be gained by a review of conclusions from time to time. Longer experience alone can tell how lasting the good results will be and how great is the danger of further scarring and narrowing of the valve.

Contraindications or Unfavourable Features.—Hypothetical speculation may well confuse these two, and it is important to distinguish between them. Moreover, what now seem to be true contraindications may later be shown to be only unfavourable features. At present the contraindications are few: among these we would include rheumatic activity that can be recognized clinically, severe disease of the aortic valve, severe mitral incompetence, severe tricuspid stenosis, a patient who is too old, perhaps over 55 years of age. We would add irreversible pulmonary hypertension or a heart muscle that is irretrievably damaged, but at present there are no means of being sure of either. Unfavourable features, which are certainly not contraindications, include a large heart, moderate aortic valve disease, some degree of mitral incompetence, extensive calcification of the mitral valve, functional tricuspid incompetence, auricular fibrillation, congestive heart failure, a history of embolism, and considerable pulmonary hypertension. Each of these is discussed below.

Age and Sex of Patients Operated On

Of the 100 patients 30 were men and 70 women. The ages ranged from 19 to 53; 12 were under 24 and 10 over 45 years of age. Table II shows the age incidence, separately for each sex and for those with and without normal rhythm: 78 of the patients were between 25 and 45, 47 of these being between 30 and 40.

TABLE II.—*Age and Sex Incidence of 100 Patients Submitted to Mitral Valvotomy*

	Age in Years							
	−19	20–24	25–29	30–34	35–39	40–44	45–49	50–54
Men (30) ..	1	0	5	4	10	8	2	0
Women (70) ..	2	9	11	16	17	7	4	4
Total (100) ..	3	9	16	20	27	15	6	4
With auricular fibrillation (44)	—	3	2	5	14	12	4	4
With normal rhythm (56) ..	3	6	14	15	13	3	2	—

We suggested in our first paper that it was doubtful if operation should be done below the age of 17, and that great caution must be used up to the age of 20. This is because of the greater danger that active, even if latent, rheumatic carditis may interfere with the outcome; but, if the mitral stenosis is so severe that it is threatening life, the age should not stand in the way. Experience shows that operation may become imperative in the late teens or very early twenties. The first of two patients aged 19 had attacks of pulmonary oedema which threatened her life and recurred as soon as she left the shelter of hospital (B19); several features suggested recent active rheumatism, but it was thought that valvotomy must be done even if it should need repeating in later years.

A patient may, however, sustain severe valve damage from rheumatic fever early in life and develop grave symptoms in late adolescence without any evidence of recurrent or smouldering disease. Thus a girl aged 19 who had acute pulmonary oedema gave no history of rheumatic fever since her first attack when 7 years old (G40). Yet at operation the valve orifice was very small and fibrous, with a craggy mass of dense calcification on the medial commissure; once the commissures had been divided the flaps were quite thin

and pliable. The whole appearance was like that commonly seen in patients 20 years older, and strongly suggested that this severe mechanical derangement had followed the attack at 7 years and that there was no special risk of recurrent rheumatism.

We have operated on four patients who were 50 years of age or over. Three have done well—two after a precarious convalescence; it is too early to assess the result in the fourth. In general we are reluctant to operate on patients as old as this, especially with limited operating resources and the long waiting-list of much younger patients in whom greater and more lasting improvement can be expected with a lower risk. On the other hand, each case demands a decision on its own merits. Some older patients may exhibit other unfavourable features such as tricuspid regurgitation, hypertension, or coronary insufficiency, all of which have been found in one or more of our patients. A case may be made out for operation as late as the middle fifties in special circumstances, but probably not beyond this.

Pulmonary Congestion

It is doubtful if we, as doctors, have appreciated how much his disability means to the patient. To be unable to hurry or climb stairs without paying a penalty, to forgo healthy physical pursuits, to have to sleep propped up in order to avoid suffocating at night, and to live in constant fear of a stroke, of haemorrhage from the lungs, of drowning in his own expectoration, or of becoming waterlogged, are just a few of his troubles. What a load is here for anyone to carry, and what a nightmare for the apprehensive. No wonder that patients seize the chance that surgery now offers.

Of course, many patients with mitral stenosis remain symptom-free for an unpredictable time, occasionally throughout a long and active life; in our experience the development of symptoms means that the disease is advanced and that stenosis is already tight. The average time from onset of serious symptoms to complete incapacity is seven years. For these reasons progressive disability in a patient under 40 with mitral stenosis provides a strong argument for valvotomy, as in such circumstances a rapid downhill course ending with acute pulmonary oedema or right-sided failure is likely. Thus operation at an early stage may be expected to save the patient much distress; it is likely to be safer, and for a variety of reasons may well prove more successful.

The various manifestations of pulmonary congestion provide the commonest and most clear-cut indications for operation, and it is here that the best results may be achieved. The most favourable type is the young patient (25–35 years of age) with a small heart and a high-grade pure mitral stenosis in whom the effects of the obstruction have fallen entirely on the left atrium and the lungs. This type was discussed in some detail in our earlier paper: the signs and symptoms cover the whole range from attacks of "bronchitis" to cardiac asthma and pulmonary oedema. In 22 of our first 50 cases severe pulmonary congestive features were the chief reason for operation; in 19 they were present but overshadowed by some other symptoms; and in 9 they were not present.

It is in pulmonary oedema that operation is particularly urgent, and this was so in 6 of our 50 cases. Attacks seem to be more frequent during the winter, presumably because of the greater incidence of respiratory infections. The problems of pulmonary oedema are so important that this condition is dealt with in a special section later.

Size of the Heart

Generally in rheumatic heart disease the size of the heart varies with the stage and the severity of the condition: the larger the heart the worse the prognosis is current and justified teaching. Gross enlargement in mitral stenosis may therefore indicate a condition too advanced for operation or unlikely to benefit as much as patients with smaller hearts. Table III shows, however, that this one factor

TABLE III.—*Results of Mitral Valvotomy According to Heart Size*

Cardiothoracic Ratio	No.	Good	Fair	Poor	Died
-50	5	4	0	1	0
50–54	19	11	2	4	2
55–59	13	8	2	0	3
60–64	11	8	0	1	2
65+	2	1	0	0	1
Up to 54 ..	24	15	2	5	2
55 and above ..	26	17	2	1	6

should not be given too much weight if other features are favourable. It is true that four of the five patients with a cardiothoracic ratio of under 50 have reached the highest grade of improvement, the fifth having mitral incompetence. Yet among the patients with the larger hearts there have also been excellent results; two with ratios of 64 and 61, both with congestive failure and totally incapacitated, have been at full work for over two years since their operation; two others with tricuspid incompetence and ratios of 62 and 63 are notable successes, and two with ratios of 68 and 70 (one operated on since the first 50) both did very well.

If the cases are divided into those with cardiothoracic ratios above or below 55% they fall by chance into two almost equal groups. It is true that the death rate is three times as high in those with the larger hearts; there are, however, as many good results in these, as is clearly shown in the last two lines of Table III. The great improvement may not prove as lasting in those with the larger hearts, and ideally they should be given the chance of operation at an earlier stage. It may be that we have applied more rigorous criteria of selection to those with the larger hearts, but this is not certain. It is clear from these figures that considerable enlargement is not a contraindication to operation, though it is generally a sign of more advanced disease that should be carefully analysed and weighed.

Nor does absence of enlargement always indicate a good prognosis. The patient with extreme mitral stenosis and a relatively good myocardium may have no enlargement and yet may have intense pulmonary congestion. In such patients the significance of bronchitis or haemoptysis may be missed and, even though the diagnosis of mitral stenosis has been made, paroxysmal dyspnoea may be mistaken for bronchial asthma or even for neurosis, especially when the pulmonary congestion has been increased by emotional causes. Such a mistake may prove disastrous, and it is therefore important to emphasize that a patient can die from mitral stenosis with a heart that is little if at all enlarged. In this type operation is very successful.

The dilated chamber or chambers chiefly responsible for the cardiac enlargement can usually be recognized by physical examination with radioscopy and with electrocardiography. Most of our cases showed right-sided enlargement which varied in degree from slight to severe; gross right-sided enlargement was generally associated with tricuspid disease (see below).

Patients with conspicuous left ventricular enlargement were not treated surgically, on the ground that aortic disease, mitral regurgitation, or systemic hypertension was mainly responsible for the disability. In the patients with such complications who were submitted to valvotomy the left ventricle was little if at all enlarged; this was so in five patients with aortic disease, in seven in whom a significant degree of mitral regurgitation was suspected, and in two with hypertension.

The left atrium was slightly or moderately enlarged in all 50 cases—often only slightly in those with the most severe symptoms. No patient with gross or aneurysmal dilatation was selected for operation, on the ground that such dilatation usually means predominant mitral regurgitation rather than stenosis. If it is reasonably sure that the dilatation is associated with severe mitral stenosis, operation is not contraindicated, though perhaps the surgeon may have to contend with a large amount of laminated clot.

Aortic Stenosis and Incompetence

When a patient has aortic and mitral valve disease it is generally possible to assess which is the more important. The character of the pulse and the pulse pressure are a guide to the degree of aortic involvement, and if there is left ventricular preponderance in the electrocardiogram and left ventricular enlargement on radioscopy these will indicate that aortic disease is of major importance. If this is not so, and the patient is otherwise suitable for mitral valvotomy, the mere presence of aortic stenosis or incompetence, or both, should not prevent operation. That the aortic valves are not normal seems no reason why the left ventricle should be starved of blood. If, however, these signs indicate that aortic disease is the more important, even successful relief of mitral stenosis is unlikely to give enough improvement to justify operation. Later, when the surgery of the aortic valve has advanced further, it may be possible to carry out a double operation on both valves.

Five of the 50 patients certainly had disease of the aortic as well as of the mitral valve. In a few others a soft basal diastolic murmur was heard but might have been due to pulmonary regurgitation. In one woman with clear signs of aortic stenosis and regurgitation the extreme disability seemed mainly due to mitral stenosis; she died, as the result of operation, from damage to the papillary muscle controlling the aortic cusp of the mitral valve (G12) (Brock, 1950): necropsy showed that the aortic lesion was not gross, and the result would probably have been successful had she survived, though there was a tendency to left ventricular preponderance in the electrocardiogram. Four other patients had clear but lesser signs of aortic stenosis or incompetence. The result in one is excellent, and operation has enabled her to return to a full life; in two the result is good, but in one it is only fair. In two of these patients the murmur of aortic stenosis increased greatly and a thrill was felt for the first time—striking evidence of the increased flow of blood through the left ventricle following the relief of the mitral stenosis. In none of these five cases with aortic disease did we regret the decision to operate, but in all there was evidence that the mitral stenosis was the main cause of the patient's disability.

Mitral Incompetence

A small rigid orifice, especially if calcified, is bound to remain partly open in ventricular systole and allow some reflux of blood. Often in such cases valvotomy can restore almost normal valve function; in quite a number we have found the cusps, outside the rigid orifice, thin and supple. The valvotomy, in addition to relieving the stenosis, can thus restore some degree of normal movement and suppleness to the valve-flaps, allowing more efficient closure. The unfavourable type is the one found at operation to have an orifice much larger than usual in "pure" mitral stenosis and with rigid resistant cusps. It is doubtful if any attempt at valvotomy should be made if this type is found at operation, but perhaps a method of relieving the incompetence will be devised later.

Mitral incompetence may be only an unfavourable feature, or it may be an actual contraindication to valvotomy. Its recognition is therefore of great importance, but is often far from easy. We have relied chiefly on three major criteria: clinical, electrocardiographic, or radiological evidence of enlargement or increased pulsation of the left ventricle in the absence of aortic incompetence; a loud mitral systolic murmur, with or without thrill; and considerable systolic expansion of the left atrium as seen fluoroscopically in the antero-posterior or first oblique position. It is also agreed that aneurysmal dilatation of the left atrium is usually associated with mitral incompetence rather than stenosis. These signs are not completely reliable, as our experience shows.

When these signs have been marked we have not advised operation, but it is important not to deprive a patient of the benefits of mitral valvotomy by overestimating the significance of any regurgitant element. When these signs have been moderate they have certainly not prevented successful and even dramatic results: they have not, however, always been confirmed by the surgeon's assessment at operation.

In 11 patients mitral incompetence was suspected clinically and confirmed at operation: in seven of these the results were good, in two fair only, and in two poor. In eight of these calcification was present, including the four in whom the results were not good.

In two patients with clinical evidence of mitral incompetence the valve was no different at operation from many others in whom "pure" stenosis had been diagnosed and did not show calcification.

A marked regurgitant stream was found in five cases in which mitral incompetence had not been diagnosed before operation. The results were good in three, fair in one (B4), and poor in one (G33), though in two there was a good deal of calcification.

Until more exact methods of assessment are available it seems inevitable that at times a case of predominant mitral incompetence will be explored in error: in one case (G36), not included in this analysis as it was not among the first 50, it was clearly impossible to improve the function of the badly damaged valve by valvotomy, and the patient died.

Calcification of the Mitral Valve

Although it has been suggested that a calcified valve is not amenable to division, this is not borne out by practical experience. Calcification of the valve was present in 40 of the 100 and in 19 of the first 50 cases: all the more severe examples had been noted before operation. It varies considerably in degree, significance, and type, and it is not entirely satisfactory to group all cases together as if the condition were an entity. Thus it may be a fine surface deposit, or a hard craggy exuberant mass may completely replace a part of the valve; in others flat plaques lie within the substance of the valve; yet again the calcification may extend out as far as the wall of the atrium and even encroach upon it; calcification of such an extent must clearly cause very grave interference with the valve function, in contrast with the first type. Light surface calcification was present in four patients, of whom two died and two gave a good result. In the other 15 calcification was heavy; one died, in seven the results were disappointing, and in seven they were good.

In the valve that was most extensively calcified the commissures split rapidly and easily, rather as an egg-shell breaks (G14, Fig. 1). In several others a hard exuberant mass of calcium in one commissure (most commonly the

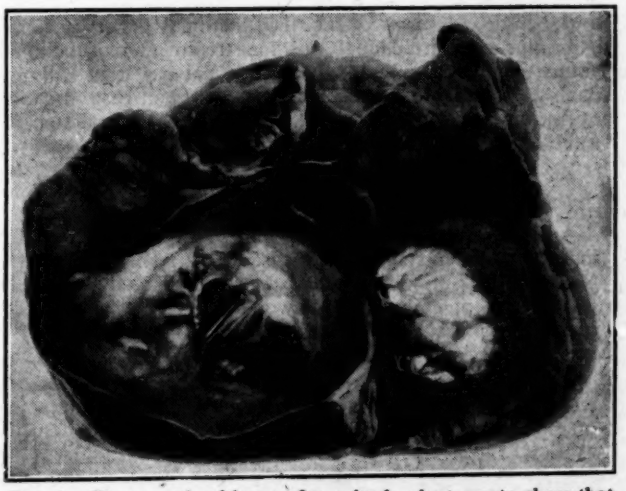

FIG. 1.—Photograph of heart after mitral valvotomy to show that an almost normal-sized orifice has been restored; note that the opening has not been merely dilated. Extensive calcification was present.

medial) either prevented or limited division in that direction, although the opposite commissure was always split or cut. Only once, when dense calcification surrounded the whole of the tiny orifice and extended for over a centimetre along the matted chordae tendineae, was division difficult; forcible digital dilatation was alone possible (G25).

In only one case was embolism, and that of a minor nature, recognized to occur after division of a calcified valve, even though in some the calcified area was felt to be crumbling and friable.

The annulus fibrosus was noted radiologically to be calcified in one case (G16), and this was confirmed at operation. During the manipulations of valvotomy the calcified ring fractured and somewhat free haemorrhage occurred from the auriculo-ventricular junction. This ceased after simple finger pressure for a few minutes; a small pack of gel-sponge was laid over the area before the pericardium was closed. The patient recovered.

A good result was obtained in only 9 of the 19 patients with calcification—a smaller proportion than in those without it. The presence of calcification of the valve seemed to be chiefly responsible for the lack of success in only 3 of the 10 who did not do well.

Tricuspid Stenosis and Incompetence

Two patients presented with the classical features of severe tricuspid incompetence, but after prolonged medical treatment they suddenly lost the characteristic systolic venous pulse; it was then realized that the incompetence was functional.

The first (B9) was a man aged 22, the second (B14) a housewife of 51 who also had essential hypertension; both had been incapacitated for over four years, both had fibrillation, and both had been in congestive failure, the woman with gross ascites; both had a large heart with cardiothoracic ratios of 62 and 63; both had pulmonary congestion and pulmonary hypertension (mean pressures, 47 and 55 mm.). Following operation the man has returned to clerical work and the woman has no difficulty in walking a mile (1.6 km.). (Since this series was completed there have been four other patients with functional tricuspid incompetence of short duration, in whom operation was advised with no hesitation.)

A third patient in the series (G34), when seen six months after operation, showed signs of tricuspid incompetence, despite very great functional improvement. The fourth (G3), reported in our earlier paper, had advanced disease and did not survive operation; necropsy showed a moderate degree of tricuspid stenosis not diagnosed in life.

We are not suggesting that every patient with tricuspid incompetence is suitable for operation, but if it is functional there is at least a case for it. It may be difficult to decide if the tricuspid incompetence is functional or organic. If it disappears with adequate medical treatment its functional nature can be assumed. Even if it persists it is still not necessarily organic, as was shown by a recent case in which operation was withheld but at necropsy tight mitral stenosis was found with a stretched tricuspid orifice.

Auricular Fibrillation

Auricular fibrillation is unfavourable in the sense that it indicates a later stage of the disease. In addition, thrombosis in the auricular appendage is more likely, with the greater risk of embolism. It would, however, be wrong to count the presence of fibrillation against operation, as it was present in 44 of our 100 patients.

In these 100, most of those with auricular fibrillation were over 35 years of age (nearly 90% over 30) and most of those with normal rhythm were under 40 (91%). On the average the former were nearly eight years older (39.1 against 31.6), and the difference was no greater because so many in each group were between 35 and 39 years (see Table II). Fibrillation was always present in those with the larger hearts, but sometimes in those with a small heart, so that the average cardiothoracic ratio was only a little greater in those with fibrillation (57.6 compared with 55.1). Among the 44 with auricular fibrillation there were 8 deaths—4 from embolism—while in the 56 with normal rhythm there were 5 deaths (18% compared with 9%). These figures bear out what might be expected—that fibrillation not only indicates a more advanced stage but adds to the operative risk from embolism.

Auricular fibrillation is in no way a contraindication to operation even if embolism has already occurred; in fact, the onset often indicates the need for a decision. In one of the early patients (G5) with increasing dyspnoea, who was being considered for valvotomy, the onset of fibrillation precipitated right-sided failure and later on attacks of cardiac asthma; it was thought at the time that operation had been delayed too long, but after two years the result remains excellent. Ideally, the patient who is losing ground should be given the chance of operation before fibrillation has become established; even fibrillation for some years does not preclude operation, but it does increase the risk, especially of embolism.

In several cases auricular fibrillation developed while the patient was under observation or while waiting for operation. Auricular fibrillation may develop after operation, and this occurred between the first and fifteenth days in 18 of the 56 in normal rhythm in our first 100 cases; in half this was temporary, normal rhythm recurring spontaneously or with quinidine; one of these later went into permanent fibrillation.

Congestive Heart Failure

Thirteen of our 50 patients had been in right-sided failure; all except one had fibrillation, and in most of them the onset of arrhythmia had precipitated congestive failure, as happens so often. In 5 of the 13 right-sided failure had been noted for at least two years, the extreme being four years; each had a large heart with a cardiothoracic ratio from 57 to 68 (average 63.6); one died, in one valvotomy was not possible, and in three the result was good.

In the other eight, right-sided failure had been present for from 3 to 12 months, and this was often the reason for advising or expediting surgical treatment; the average cardiothoracic ratio was 58.5. One of the eight died; the result was poor in one, fair in one, and good in five of these patients.

Two out of the eight deaths, and three disappointing results out of the nine in the series of 50, is not a high proportion, and shows that right-sided failure is no bar to operation. Certainly the eight patients who benefited showed very gratifying results, and two have continued at work for over two years since the operation. It must be remembered, however, that they were selected from many others with congestive failure, in whom the advanced stage of the disease appeared to be an absolute contraindication, and made it unlikely that they could have stood either anaesthetic or operation. Mitral valvotomy should not be considered the last resort of the dying, but right-sided failure, if not of long duration, and particularly if it responds quickly and can be controlled by medical treatment, is no bar to operation when the patient is otherwise suitable. It would therefore appear that operation may be justified in some apparently hopeless cases, at any rate until greater experience has supplied more certain information.

Embolism

It was thought at first that a history of previous embolism introduced the extra hazard of further embolism occurring as a result of operation. Experience has not supported this view, and we now regard such a history as one of the indications for valvotomy in the hope that less stasis in the atrium will discourage clotting.

Previous embolism had occurred in 17 of our 100 cases, five while in normal rhythm, but at operation in eight of these no thrombus was found in the auricle. A history of embolism

therefore does not necessarily mean a thrombus that may be disturbed. In contrast a thrombus (old or recent) was found in 21 cases with no history of embolism; in 13 of these the removal of a recent soft clot may well have prevented an embolism occurring. In one case (B2) extensive thrombosis extending on to the adjacent atrium made it unsafe to open the heart.

Embolism occurred after operation in seven patients. Of these, four died, one having had a previous hemiplegia. In this patient (G11) the wall of the atrium was found *post mortem* to be covered with a thick irregular layer of clot of varying ages over an area of some 75 cm.[2]; an embolus was found in the right middle cerebral artery. A second patient (G2), who had a partial hemiplegia, died from massive haemothorax following too early use of heparin. A third (G57), with a history of two previous emboli, had both old and fresh clot in his auricular appendage, did not recover consciousness after operation, and died from cerebral embolism. In the fourth (G17), a man aged 39, the appendage was seen to be small and completely obliterated by firm organized clot. By patient dissection a large polypoid mass of this was freed, but a further fragment of clot was felt momentarily in the atrium before the valve opening was enlarged. Soon after this the anaesthetist reported an unusual rise in blood pressure to 158/110. Before leaving the theatre the patient complained of severe pain in his legs, which he did not move although he was flinging his arms about. Examination disclosed an aortic embolism, with no pulsation below the umbilicus. Anaesthesia was reinduced, and a large saddle-embolus removed from the aortic bifurcation; the circulation in the lower limbs returned at once. He made a good immediate recovery, and the next day seemed none the worse except for pins-and-needles in the soles of his feet. Unfortunately he suddenly became unconscious 36 hours after operation and died after a few hours. At necropsy a small embolus was found in the basilar artery; the site of the aortic incision was quite healthy.

Since that case all the accessible large arteries are carefully palpated before operation and before the patient leaves the operating theatre; the significance of an unusual rise in blood pressure is also appreciated. In addition, if old or new clot is found in the auricle or if suspicious induration is present the auricle is incised and allowed to gush blood for a moment before the clamp is applied, or the clamp is momentarily released to allow a rush of blood to wash out any contained clot. This manœuvre has been successful in clearing clot in seven cases; in one a large clot fragment some 1.5 by 1.5 cm. was projected on to the floor near the operating table. There is therefore no doubt that this is an important practical manœuvre. Post-operative embolism occurred 4 times in the first 24 cases with 3 deaths, and 3 times in the last 76 cases with one death. We do not use heparin as a routine.

Haemoptysis

Haemoptysis is common in mitral stenosis and varies in degree from the blood-flecked sputum that so often accompanies acute pulmonary congestion to massive unexpected haemorrhage. Blood-streaked sputum accompanying congestive attacks is always less important than the distress caused by cough, dyspnoea, and orthopnoea; it occurred in two-thirds of our cases and did not influence the clinical assessment. In other words, an attack of "bronchitis" or of acute pulmonary congestion is neither more nor less serious because of the presence or absence of blood-flecked sputum. The prognosis in these cases is also the same whether slight haemorrhage accompanies the episodes or not. Similar observations apply to acute pulmonary oedema, the attack being neither more nor less dangerous on account of the presence or absence of *pink* frothy sputum. A third form of haemoptysis in cases of mitral stenosis results from pulmonary infarction. This is usually a late event and tends to complicate congestive heart failure, the infarct being due to an embolus liberated from phlebothrombosis in the veins of the legs or from the right atrium. The clinical importance of such haemoptysis depends on the size or number of the infarcts; the prognosis may be adversely influenced by the tendency to thrombo-embolism, but is determined chiefly by the heart failure itself.

These well-known facts have been mentioned in order to avoid confusion, because the haemoptysis we wish to discuss is a fourth type—the sudden frank haemorrhage that may be termed pulmonary apoplexy. This usually occurs unexpectedly when the patient is otherwise well, and may be the first symptom of the disease. Such haemorrhages are often massive—½ to 1 pint (284 to 568 ml.)—but do not last long, rarely more than 24 to 48 hours. In our first 50 cases 20 had haemoptysis of this kind. In 12 of them only one frank bleed occurred, in 4 there were two such haemorrhages, and in the remaining 4 severe recurrent haemoptysis dominated the whole clinical picture.

It is by no means uncommon for a patient to give a history of one or two attacks of pulmonary apoplexy many years before more serious symptoms develop. Precipitating factors include pregnancy and unaccustomed effort, but the trigger is often obscure. Sometimes repeated haemoptysis ceases after a year or so, and does not recur later when the disease is more advanced. There is little evidence that this course is due to diminished pulmonary congestion resulting from active pulmonary hypertension, right ventricular failure, or tricuspid incompetence, although such complications must protect the pulmonary capillaries and venules to some extent; again, haemoptysis may not recur when signs and symptoms of pulmonary congestion are obviously progressive. It is suggested that frank haemoptysis is more likely to occur when the pulmonary venous system is unaccustomed to working at a high pressure, and may cease when the vessels have adapted themselves to the situation. It is known that veins thicken quickly when subjected to a high internal pressure. The point to be emphasized is that pulmonary apoplexy is not necessarily a bad omen, and is rarely an indication for urgent valvotomy. In this respect it is far less serious than orthopnoea and paroxysmal cardiac dyspnoea, and is not to be compared with pulmonary oedema. Moreover, a good bleed relieves pulmonary congestion, and is therefore self-limited. On the other hand, the effect of valvotomy on the tendency to haemorrhage from the lungs is quite remarkable, for there has not been a single haemoptysis of any kind after a successful operation.

Haemosiderosis

Skiagrams revealed unquestionable haemosiderosis in 5 of the first 50 cases. This could not be correlated with any other common factor. There were three men and two women, and their ages ranged between 26 and 40 years. Two had had repeated severe haemoptyses, but two had never coughed blood at any time. Four had normal rhythm, but one had fibrillation. The mean pulmonary artery pressures were relatively low in two (25 and 27 mm. Hg), moderately raised in two (50 and 53 mm. Hg), and very high in one (100 mm. Hg). Four had practically pure mitral stenosis, but the most severe example had moderate incompetence as well. The results were excellent in two cases, good in one, fair in one, and one patient died from cerebral embolism. Subsequent skiagrams showed no change in the characteristic peppering in the four who survived. It is concluded from these and other similar cases that haemosiderosis should not influence the decision to operate one way or the other.

Pulmonary Hypertension

When the left atrial pressure rises, the pulmonary artery pressure must follow suit if the proper gradient between the two is to be maintained. The pulmonary capillary pressure —measured distal to a pulmonary artery branch that has been blocked by the cardiac catheter—usually represents the pulmonary venous pressure and gives a rough estimate of the left atrial pressure. Cases of mitral stenosis with minimal symptoms have usually shown a pulmonary capil-

lary pressure of about 10 mm. Hg and a mean pulmonary artery pressure of about 20 to 25 mm. Hg. Most patients submitted for operation have had a resting capillary pressure of 20 to 30 mm. Hg and a mean pulmonary artery pressure of 30 to 50 mm. Hg, giving a similar gradient of 10 to 20 mm. This may be called passive pulmonary hypertension.

When the pulmonary capillary pressure is around 35 mm. Hg pulmonary oedema may be expected, so that persistently higher left atrial pressures are incompatible with life. In fact, we have not recorded a resting pulmonary capillary pressure much above this.

A group of cases of some interest are those with resting mean pulmonary artery pressures at least 30 mm. Hg above the pulmonary capillary pressure, or of at least 60 mm. Hg if the latter has not been measured; here the gradient between pulmonary artery and approximate pulmonary venous pressure is well beyond the limits of passive pulmonary hypertension, and implies a true increase of pulmonary peripheral resistance on the arterial side of the capillary bed. Twelve of our first 50 cases had this greater degree of pulmonary hypertension, 10 of the 34 at Guy's and 2 of the 16 at Brompton. Selection is likely to have played an important part in determining so high a proportion in the Guy's series, for in a series of 88 cases investigated at Brompton and at the Institute of Cardiology active pulmonary hypertension was encountered in only 11 cases.

These cases can usually be recognized clinically by a variety of signs that cannot be discussed here. The present problem is how they fare after valvotomy. The results did not differ from the series as a whole. Of the 12 cited, five did excellently, three well, two poorly, and two died. Catheterization was repeated 6 to 12 months after operation in six of them, and in the five who were much improved clinically the pulmonary artery pressure had fallen to half or even to a third of its previous level (Table IV). The

TABLE IV.—*Comparison of Results of Catheterization Before and After Mitral Valvotomy*

Case	Mean Pulmonary Arterial Pressure (mm. Hg)*			
	Rest		After Exercise	
	Before Operation	After Operation	Before Operation	After Operation
G1	64	32	—	—
G4	40	27	—	—
G5	46	22	—	42
G6	100	55	116	79
G8	85	24	—	42
G9	52	35	75	44
G10	71	30	115	36
G13	71	22	109	48
G27	57	36	—	52
G31	53	23	104	44
G34	33	15	32	—
Unsuccessful Cases				
G16	100	88	115	141
G25	42	67	—	79

* Measured from a point midway between front and back of chest, at the level of the fourth costal cartilage.

sixth case was not clinically improved by valvotomy, and recatheterization showed no appreciable fall in the pulmonary blood pressure.

Of these 12 patients five had a resting pulmonary systolic blood pressure above 100 mm. Hg. It might be suspected that such pressures would prove irreversible, so the results in these five are exceptionally interesting. Two did excellently, one well, one poorly, and one died. Catheterization in the first two revealed a profound fall in pulmonary blood pressure, from 130/78 to 68/42 in one (G6) and from 130/56 to 40/10 in the other (G8); the third patient who did well (G29) has not yet been recatheterized.† The one who did poorly (G16) had a pulmonary blood pressure of 134/67 before valvotomy and 142/69 some eight months later; the operation in this case was not technically satisfactory. It is concluded that even severe pulmonary hypertension up to 130/80 mm. Hg is not necessarily irreversible. Indeed, we have no example in mitral stenosis of irreversible pulmonary hypertension, a term that can be applied only when the pressure is not materially lowered by a technically successful mitral valvotomy. Nevertheless, we should be surprised if this situation was not encountered sooner or later. In the meantime we have shown that severe pulmonary hypertension must not be regarded as a contra-indication to surgical treatment.

†Post-operative catheterization has since shown a mean P.A. pressure of 23, the original pressure being 84 mm. Hg.

Acute Pulmonary Oedema

Acute pulmonary oedema is so important that it demands special consideration; its full significance in mitral stenosis and its correct management are not yet fully appreciated.

The mechanism of the formation of oedema is some final disturbance of the very delicate balance that exists between the hydrostatic and osmotic pressures in the pulmonary capillaries. The obstruction to the emptying of the left atrium causes a rise in pulmonary venous capillary pressure and a secondary passive rise in pulmonary arterial pressure.

The increased pressure gradient across the mitral valve serves to maintain proper left ventricular filling, and therefore an adequate cardiac output. This mechanism has two serious drawbacks: (1) sudden elevation of the pulmonary venous pressure causes reflex dyspnoea (orthopnoea and paroxysmal cardiac dyspnoea); and (2) if the pressure head rises above about 35 mm. Hg the hydrostatic pressure in the pulmonary capillaries may exceed the osmotic pressure, so that pulmonary oedema results. Attempts to raise the cardiac output in accordance with physiological needs are initiated by adjustments such as tachycardia, a rise of systemic venous pressure, and increased strength of right ventricular contraction. The spate of blood poured into the lungs cannot escape through the mitral valve without a sufficient rise of pulmonary venous pressure. This increases the pulmonary peripheral resistance, and the pulmonary blood pressure rises as already stated. A weak right ventricle may fail to deal with the added load, and its output then tails off: this may save the patient from pulmonary oedema at the expense of an inadequate cardiac output for the occasion. Weakness, dizziness, or a feeling of exhaustion then brings the effort to an end; the stimulus ceases, the right ventricular output returns to normal, and the pulmonary spate subsides. When the right ventricle is powerful, however, it may be able to deal with the increased load and may continue to pour blood into the lungs against an ever-increasing resistance. In such circumstances the only physiological event that can save the patient from acute pulmonary oedema is peripheral pulmonary vasoconstriction, so that the pulmonary arterial pressure rises far beyond the level conditioned by the rise in pulmonary venous or capillary pressure. Even the strongest right ventricle may have difficulty in surmounting this obstacle.

It follows that pulmonary oedema is most likely to occur in patients with tight mitral stenosis who are equipped with a powerful right ventricle and with sensitive and energetic reflex adjustments for raising its output, but have little tendency to develop protective pulmonary vasoconstriction.

Isolated attacks of pulmonary oedema or severe cardiac asthma are often found in the history. They are common in pregnancy, and may follow an acute respiratory infection, extra fatigue or effort, or emotion. Several of our patients have told of attacks on the way home or on arrival home after a late dance. Sexual intercourse may also be a cause. The journey to hospital may well precipitate an attack. Several patients have been in early oedema in the out-patient department and have needed immediate admission; one died less than two hours later.

We have observed rapidly occurring mild oedema with the preliminaries of cardiac catheterization, or even when the patient is approached for consultation. The disturbances preparatory to operation may be much more serious; two patients developed severe acute pulmonary oedema in the anaesthetic-room just before induction; clearly the

greatest care must be taken to lessen the risk of this, especially by adequate early sedation and by avoidance of all fussy preparation in the last hour or two of waiting: an orthopnoeic patient should not be laid flat during this period.

Early isolated attacks usually subside spontaneously, but later, when a state of continued and severe pulmonary congestion is present, an attack may prove fatal unless operation can give relief. In such cases pulmonary oedema is an emergency calling for prompt and vigorous first-aid treatment—an emergency just as important in its way as mechanical obstruction of the air passages or of any hollow viscus.

Treatment of an Acute Attack

A mild attack may be short and may respond readily to simple treatment, but the course of an attack is uncertain and may prove rapidly fatal when all seems improving. A vicious circle is apt to be set up by the patient's distress and exhaustion, and death occurs from inability to clear the air passages, the patient drowning in the fluid.

Treatment should be begun promptly and vigorously. The patient should *not be moved until initial treatment at least has been given.* He should be propped upright with the legs dependent, preferably in a simple wooden chair. Venous tourniquets should be applied immediately as high as possible on each thigh, to diminish the venous return; this serves as a rapid and simple venesection. Morphine ¼ gr. (16 mg.), should be injected intramuscularly or even intravenously; in the later stages, however, when the patient is moribund and the respiration is failing, morphine may be dangerous. Aminophylline, 0.24 g. intravenously, is of real value. Oxygen should be given, preferably with a mask. If rapid improvement does not follow these measures, venesection of 500–750 ml. should be done.

The other step of great importance is to clear the air passages by suction, because simple suffocation from drowning is the usual mode of death. A suction catheter should be passed into the trachea, either by the nose or by the mouth, or, if facilities are available, direct bronchoscopic suction should be used. As soon as it is realized that a severe attack has begun the simple instruments for this suction should be prepared; they should be used as soon as there is any suspicion that the patient is failing to cope with increasing fluid in the air passages. It is usual to err on the side of waiting too long. The development of thoracic surgery and anaesthesia during the past 15 years has made us increasingly conscious of the great value of suction in clearing the air passages in many conditions; its importance in the treatment of acute pulmonary oedema should be widely realized, as it may be life-saving.

Treatment of Recurrent and Relapsing Pulmonary Oedema

A patient may have one or more isolated attacks of acute pulmonary oedema over several years, perhaps caused by a special strain or effort. With added care and avoidance of similar stress there may be no recurrence. When, on the other hand, attacks are frequent or when there is a state of chronic pulmonary congestion with frequent exacerbations the time has come to cut short the process by valvotomy if the patient is to survive. The need for operation is urgent, and we have learned to have great respect for this state of affairs.

The congestion should first be lessened by routine medical treatment, but the resulting improvement should not lull the physician into a sense of false security, and operation should not be postponed; indeed, *the patient should not be allowed to leave hospital until it is done.* This is equally the case if routine treatment fails or leads to only slight improvement; only in this way is the patient's life likely to be saved. Valvotomy was undertaken urgently in nine such patients, and the following three illustrate this policy.

Case B13.—A man aged 28 was found to have mitral stenosis on discharge from the Navy in 1947. He was then given a good prognosis by a cardiologist, but two years later began to notice an increasing disability, culminating in large haemoptyses in 1951, and he then had to give up work. When first seen by us he had severe pulmonary congestion clinically and radiologically and moderate pulmonary hypertension (56/32 mm. Hg). Operation was advised, and he was sent home to await admission, but one week later he had to be admitted to another hospital as an emergency. One week later he was transferred to the Brompton Hospital, but arrived almost moribund in severe acute pulmonary oedema. In spite of strict medical treatment his condition remained precarious; intense congestion was present all the time and it was clear that, if he was to survive, operation had to be done in spite of the obvious risk. He made a good recovery from valvotomy and has since returned to full-time work with no symptoms whatever; all pulmonary congestion has been relieved.

Case G32.—A man aged 42 developed severe pulmonary oedema following cardiac catheterization; his condition was grave for 36 hours. In view of this it was thought to be unsafe to send him home to await operation. Although he seemed well controlled he developed another attack on his way to the operating theatre and was gravely ill. He improved after energetic treatment in the anaesthetic room, and it was decided to proceed at once with the operation. This went satisfactorily and he made a good recovery.

Case B22.—A woman aged 30 was admitted to hospital in a state bordering on acute pulmonary oedema and improved on routine treatment. Cardiac catheterization revealed a pulmonary artery pressure of 90/45 mm. Hg. Ten days later, while still awaiting operation, she developed a very severe attack of acute pulmonary oedema; this improved at first with energetic treatment, but over a period of 48 hours frequent severe relapses occurred, and her condition became so desperate that it was clear she would not live unless an operation could help her. In spite of the grave risk, our experience with other severe cases encouraged the attempt. Soon after the heart was exposed ventricular tachycardia developed; she improved following a pause. Valvotomy was followed by cardiac arrest; massage was begun with no response, and adrenaline was injected. The heart began beating, but ventricular fibrillation soon appeared. This was corrected by injection into the left atrium of 10 ml. of 1% procaine solution, once repeated. She left the theatre in fair condition and in normal rhythm, but died 36 hours later with pneumonia and renal failure.

Comment.—Although Case B22 ended fatally it must be considered in conjunction with the other eight operated upon, and with success, in a similar state of severe chronic or recurrent pulmonary oedema. The condition is one of the utmost gravity, and our plea is for recognition that the condition calls for valvotomy as soon as possible.

Although the parallel may not be entirely acceptable it seems reasonable to compare the state of affairs with the more familiar one seen in obstruction of other muscular viscera, such as the bladder, stomach, or intestine. In all of these, once obstructive symptoms occur, the sooner that operative relief is given the better. Without operation the patient struggles on until virtually total obstruction occurs. Conservative measures may achieve temporary abatement, allowing operation to be done in more favourable circumstances. A stage is, however, reached at which no improvement follows non-surgical treatment, and, unless the obstruction is relieved by operation, death is inevitable. At this phase the risk of operation is much greater and the mortality of quite a simple procedure is high.

These cases of mitral obstruction also pass through the earlier phases of "subacute" or "acute obstruction" that respond to conservative measures. Operation must then be done if relapse is to be prevented. Once there is no response to conservative measures death is inevitable unless an emergency operation can relieve the obstruction; the risk is much higher, but it is a risk that must be taken. As in the surgery of obstruction of other muscular viscera, the best way to avoid high mortality is to recognize and treat the obstruction as early as possible so that a serious state of decompensation is prevented.

Pregnancy and Sterilization

It has been, and still is, a common practice in the more severe cases of mitral stenosis to terminate pregnancy and to advise sterilization; or, if the pregnancy goes to term, to advocate sterilization afterwards. This is a policy that demands reassessment in view of the present possibility of successful valvotomy. Apart from the destruction of a life by the termination of pregnancy there is the effect on the marriage itself, which may not stand the double strain of the wife's sterility and incapacity.

Valvotomy can be performed during pregnancy, which can then be allowed to proceed normally. It may be needed as emergency in the middle or later months if severe pulmonary congestion or oedema develops. Both these points should be realized by obstetricians and physicians alike.

Termination and sterilization may still be correct treatment, especially if other complicating lesions or factors are present, but often these are not justified until the exact state of the mitral valve, as revealed at operation, is known. Thus if the valve is found to be densely fibrous, rigid, and calcified, so that an adequate valvotomy is not possible, sterilization may be advisable. If the valve is found to be thin and supple so that restoration of a large valve lumen is possible, then there can be no justification for termination, and still less for sterilization.

Two of us have already reported a case (Brock and Campbell, 1950) of successful pregnancy following pulmonary valvotomy. In another case (R. C. B. and P. W.) pulmonary valvotomy was performed during the fifth month of pregnancy without event; natural delivery occurred safely.

In one case in our series of mitral stenosis valvotomy was performed successfully during the third month and the pregnancy has proceeded to natural delivery; the condition of the valve at operation justifies optimism. In this patient, who was on our waiting-list for valvotomy because of attacks of pulmonary oedema, termination of pregnancy and sterilization had actually been decided on elsewhere. In a second case a successful valvotomy was done at the 28th week in a patient who had severe pulmonary congestion with repeated small haemoptyses from the third month despite treatment in hospital. A third and almost identical case, in which termination and sterilization had been advised but was refused on religious grounds, has since had a successful valvotomy at the 28th week. Both these patients have now had a normal delivery at term.

Operative Experiences

As this paper is chiefly clinical it is not necessary to do more than mention briefly a few technical matters.

An anterior incision through the third intercostal space with division of the third and fourth cartilages was used for the first 45 cases; this gives somewhat cramped access in some, especially if there is much right-sided hypertrophy and the auricle is small. A standard postero-lateral thoracotomy has also been used but is unnecessarily elaborate; it gives an excellent exposure, but there is unnecessary interference with the big scapular muscles. We have abandoned this incision.

Our present incision is almost a straight horizontal one, skirting the lower border of the breast, curving up slightly in front and extending backwards some 3 cm. below the inferior angle for the scapula and about 10 cm. beyond it. The serratus anterior is cut at its origin; the front 10 cm. or so of the latissimus dorsi is divided. The chest is opened through the fifth interspace; if this space is incised from front to back there is no need to divide a cartilage. An excellent exposure is thus given with the minimum of muscular damage. The patient lies on the right side rotated back 45 degrees.

As described in our first paper, the valve is approached by inserting the index finger through the left auricular appendage. It is necessary to emphasize that the valve orifice is not merely stretched or dilated; it is quite wrong to describe the operation as such. The valve is actually split along the line of one or both commissures so that in a favourable case an opening of almost normal size results. It must also be pointed out that it is not necessary, in most cases, to use a valvotome for this purpose. An instrument was used in only 28 of our 100 cases. The impression has been created that without an instrument the operation cannot be done either safely or effectively. This is wrong, and in fact may be a dangerous teaching in that it encourages a surgeon inexperienced in intracardiac manipulations to make things much more difficult. In many cases the finger alone splits the valve with an accuracy and speed that no instrument could rival. It is our practice to introduce the finger and do all that is possible with it alone. If the division is not enough the finger is withdrawn and reinserted carrying a valvotome, which is a simple, thin, strip-like knife that can bend with the finger and slides to and fro through a slot in a finger ring. Quite a small incision may enable the finger to continue and complete the splitting.

The third technical matter is the management of a small contracted thrombosed auricle. The danger of embolism has already been discussed and the importance described of allowing a free gush of blood to wash away any clot. Although it is comforting to find a long patent appendage, the surgeon must not be deterred if he finds it short and solid. By careful dissection the organized or organizing thrombus can be freed and an entry made. In nine such cases in which the opening was very small and the atrial wall very thin, and it was too dangerous to insert the index finger, the little finger found its way in easily, and the valve was split with it alone in six. In the remaining three it was possible to follow with the index finger and complete the division begun by the little finger.

Cardiac Catheterization

This method of investigation has been employed in all but one of our 100 cases. The usual procedure has been to measure the right atrial pressure, pulmonary artery pressure, pulmonary capillary pressure, and cardiac output at rest, and also to measure the pulmonary artery pressure and cardiac output during a standard exercise test. Our aim in this has been not only to assist in the assessment before operation but also to gain information about the cardio-respiratory system in mitral stenosis and the changes occurring before and after valvotomy. To this end we also intend to repeat our observations after the operation. A report on such studies made on the Guy's Hospital cases is being made by Dr. Holling and Dr. Venner, to whom we are indebted for these investigations.

Cardiac catheterization is not without risk in mitral stenosis. We have had several instances of pulmonary infarction which may have been due to the wedging of the catheter into a small pulmonary artery during measurements of pulmonary capillary pressure. Very occasionally an attack of pulmonary oedema (in one case fatal) has followed cardiac catheterization. We prefer to delay operation for at least 7 to 10 days after catheterization, however well tolerated.

Studies after valvotomy in 11 cases have shown a fall in pulmonary artery pressure commensurate with the clinical improvement (Table IV). The maximum improvement may not be seen at once and our present practice is to delay catheterization until 9 to 12 months after the operation. This is shown in patient G5, whose mean pulmonary artery pressure before operation was 51 mm. Hg; six weeks after it was 42 mm. Hg at rest and rose to 62 mm. Hg on exercise. He continued to improve clinically, and when the investigation was repeated 16 months after operation the pulmonary artery pressure had fallen to 22 mm. Hg at rest and was 42 mm. Hg after exercise. This need for waiting has held up our post-operative studies, and the final assessment of the value of catheterization will not be made until they are available. It is, however, possible to give our present

opinion of its value in the investigation of cases of mitral stenosis before operation. For the most part the findings have done little more than support clinical impressions already formed, and have never caused us to alter the decision whether or not to operate. For the time being we are continuing this method of investigation in order to find out as much as possible about the changes associated with mitral stenosis before and after operation. Those who have patients clearly suitable for valvotomy by clinical criteria should not be deterred from operating because they are unable to organize the necessary facilities for catheterization.

Medical Treatment Before and After Operation

To minimize the danger of pulmonary oedema in patients awaiting operation we use sedatives and dehydrating measures, including the mercurial diuretics and a low sodium diet. Those with normal rhythm are given digitalis so that they will already have some measure of protection should they develop fibrillation after operation. At one period we also tried quinidine for some days before and after operation in an attempt to prevent this not infrequent occurrence. It was not, however, successful, and our present practice is to digitalize all patients and to use quinidine only if it is decided to try to restore normal rhythm in those who develop fibrillation after operation. The fall in blood pressure that occurs in many cases during the operation is being investigated, but the subject is too complex to discuss in this paper.

Complications after operation are few and the patient who returns to the ward after a successful valvotomy gives little or no cause for anxiety. We have had abundant experience that when the surgeon is satisfied with what he has achieved at operation the physicians have little need to worry, however ill the patient was before. The improvement to the heart is in fact the reason why patients tolerate this operation so much better than they would any other. The patient is usually ambulant at the end of a week and ready to start convalescence at the end of three weeks.

Symptoms and Signs after Operation

Symptoms are more reliable than signs as evidence of a successful result. An early and striking feature is the obvious improvement of the peripheral circulation as shown by the colour and warmth of the body and limbs and the need for fewer bedclothes. The patient is quickly aware of the improved function of the lungs and notices that "the chest feels more free," and this despite the operative trauma to the chest wall. The loss of orthopnoea is an early indication of success. The degree of functional improvement during convalescence is a fairly reliable guide to the ultimate result, though more time is needed to show whether it is to be classed as good or excellent.

The physical signs give less indication of the change that has occurred. In a few patients the diastolic murmur has entirely disappeared and in many the intensity has diminished, but the absence of any significant alteration does not necessarily mean a poor functional result. In some patients a soft apical systolic murmur has appeared or has slightly increased, but this also is compatible with a good result. More reliable indications of success are a diminution of the signs of pulmonary hypertension. The size of the heart alters little and in some successful cases not at all; we have only a few in which it is easily apparent and two in which it is striking (Figs. 2 and 3). In many, however, the contours alter, showing slight diminution of the prominence of the right border, a less bold pulmonary arc in the anterior and right oblique views, and a very slight increase in the left ventricular contour; in many there is a notable flattening of the left border in the straight view owing to the loss of the shadow of the left auricular

FIG. 2.—Teleradiographs of a man of 26 (G6) six months before and 19 months after operation. Nine years' history of increasing breathlessness and severe pulmonary congestion; he could walk only 20 yards (18 metres) without symptoms; tight mitral stenosis and marked pulmonary hypertension (mean pulmonary artery pressure 100); radiograph shows haemosiderosis. Since operation, on March 2, 1950, has led a full working life; post-operative pulmonary artery pressure, 55.

FIG. 3.—Teleradiographs of a woman of 38 (G39), the day before and five months after operation. There was six years' history of increasing dyspnoea and admissions to hospital for acute pulmonary congestion; definite but not significant aortic incompetence, auricular fibrillation for six months, and right-sided failure controlled only by complete rest (capacity D). At operation on July 12, 1951, fresh clot was washed out of the auricle and an opening of 4 cm. in length with two mobile cusps was achieved. Five months later her capacity was A.

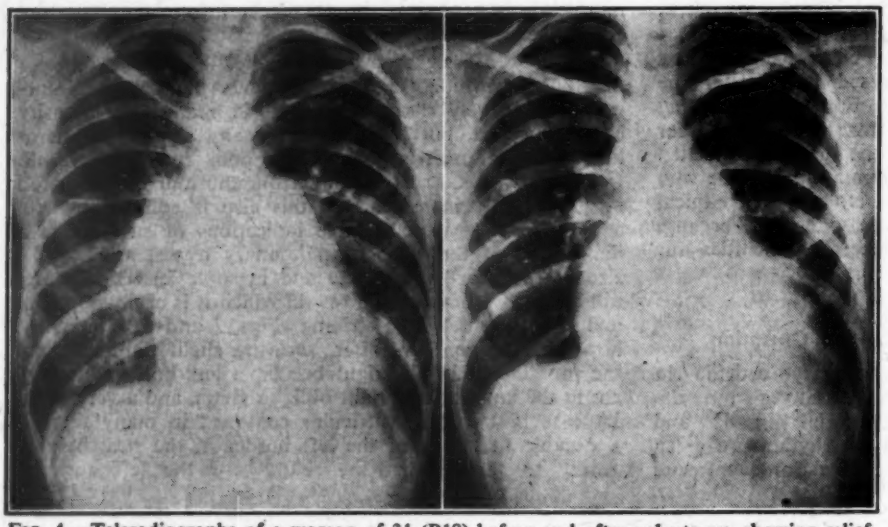

FIG. 4.—Teleradiographs of a woman of 31 (B18) before and after valvotomy, showing relief of pulmonary venous congestion. Five years' history of increasing breathlessness and bronchitis; could walk only 50 yards (46 metres); mean pulmonary artery pressure 44 mm.; successful valvotomy on May 30, 1951.

appendage after operation. Diminution of the hilar shadows denoting pulmonary venous congestion may be quite striking (Fig. 4). Though the main pulmonary vessels appear less dense and prominent in some successful cases, there is no significant alteration in the smaller vascular lung markings.

Follow-up and Future

The longest period of follow-up in the first 50 cases is 40 months, the shortest 8 months. We have deliberately not included the subsequent 50 cases in the assessment of results, though we expect that the proportion of successes in these has increased. In no case have we been far out in our preliminary judgment of success or failure, but we feel more confident of a good result when the patient has successfully withstood the advent of cold and winter weather, so often the stumbling-block or downfall of these patients with mitral stenosis, and often a more exacting test than physical exertion.

In 13 patients we have checked our clinical assessment—one poor, one fair, and 11 good or excellent results—by catheterization at periods varying from 1 to 18 months after operation, and in two patients this was done twice. In all the agreement was close.

A more prolonged follow-up is needed before any degree of permanence can be claimed, but so far the results in this respect are encouraging. The first patient operated on 40 months ago recently returned to hospital as it seemed possible that her improvement was not maintained—she was walking 2 miles (3.2 km.) instead of 4 miles (6.4 km.)—but catheterization showed no alteration from her first test shortly after operation. It is possible in the future that patients may again deteriorate either from further stenosis of the valve or from further rheumatic activity. Time alone will show, but there is as yet no evidence that such deterioration has occurred, nor should such considerations weigh against operation. Even if the disease recurs, successful valvotomy does at the least return the patient to the condition in which he was several years previously, and many of the earlier cases could not have survived till now had operation been withheld.

Summary

It is only 18 months since we reported the results in the first 9 patients submitted to mitral valvotomy. We can now report the immediate results in 100 patients, and a more detailed analysis and a longer follow-up of the first 50 of these.

The results are good or excellent in 32 of the first 50 patients. Before operation nearly all were seriously disabled : after it two-thirds have resumed work and can walk two miles (3.2 km.) or more. Often the improvement is dramatic.

There were 13 deaths in the first 100 cases ; 7 of these were in the first 20, so that for the last 80 the operative mortality has been less than 8% although many of the patients were gravely ill. The presence of auricular fibrillation, which generally indicates a later stage of the disease, adds to the risk (8 deaths in 44 cases, compared with 5 deaths in 56 cases).

Four-fifths of the patients operated on were between 25 and 45 years of age. We were soon satisfied that the young patient with pulmonary congestion, but often with a heart not greatly enlarged, could be vastly improved by operation. Wider experience has shown that increasing years or a larger heart are compatible with success.

Mitral regurgitation is not increased by a successful operation. Neither mitral regurgitation, some degree of which was present in about one-third of the patients, nor calcification of the mitral valve, nor functional tricuspid regurgitation prevents good results, but gross calcification, especially when associated wtih mitral regurgitation, does make success less likely.

No patients were included when mitral regurgitation or aortic valvular disease was thought to be the predominant lesion. Aortic stenosis or regurgitation was, however, an added lesion in 5 of the 50 patients, and this too did not prevent success.

At least 13 of the 50 had suffered from congestive failure. Good results were obtained in many of these, even when the heart was large. When auricular fibrillation and congestive failure have been present for long it seems likely that the ideal time for help by valvotomy has been missed, but their presence even for as long as five years in some patients did not prevent great improvement. Embolism was the cause of death in four of the eight patients with auricular fibrillation.

A history of previous embolism is no argument against valvotomy, and, in fact, no thrombosis was found in the auricle at operation in 8 of 17 such patients. The steps that should be taken at operation to minimize the risk of further embolism have been described.

Some cases with pulmonary hypertension may reach a stage where the pulmonary changes are irreversible, but systolic pressures in the pulmonary artery above 100 mm. Hg did not prevent excellent results. When the pressure in the pulmonary artery at rest is not very high it may rise abnormally on even the slightest exercise, with little or no increase in cardiac output. This state of affairs is often an indication for operation.

The management and treatment of acute and recurrent pulmonary oedema are discussed in detail.

Cardiac catheterization has been carried out in all our cases to obtain as much information as possible. The results have rarely changed our clinical views, and we

think that mitral valvotomy should be undertaken in suitable centres even where cardiac catheterization is not available.

Mitral valvotomy should be considered in all patients with mitral stenosis who are progressively disabled. Orthopnoea and recurrent pulmonary oedema are special indications. The risk of recurrent rheumatic carditis in patients under 20 years of age should make one more hesitant, and the presence of active carditis is one of the few contraindications to operation.

It must also be considered as an alternative to termination of pregnancy and sterilization; it has been carried out successfully in three women during their pregnancies, which have then terminated normally.

Time alone will tell how long the improvement after mitral valvotomy can last and whether fibrosis or recurrent carditis will again lead to severe stenosis of the mitral valve. In the two or three years that have elapsed since the earliest operations there has been no evidence of relapse.

We wish to thank our many colleagues who have been actively interested in the wide problems of mitral stenosis and who have helped in the investigation and treatment of these patients. The Guy's group are particularly indebted to Dr. H. E. Holling, of the Medical Research Council Unit.

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ADRENAL CORTICAL FUNCTION AT DEATH AS MEASURED BY LEVEL OF CIRCULATING EOSINOPHILS

BY

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In recent years it has been shown that the chief function of the adrenal cortex is to aid the organism in adapting to stress, either internal or external (Selye, 1949, p. 111). When an organism is subjected to stress an "alarm reaction" is set up, according to the observations of Selye. It is during the countershock phase of this reaction that the adrenal gland is called into play. There is evidence of hyperfunction of the cortex of this organ as demonstrated anatomically and physiologically. There then ensues a stage of resistance during which this hyperactivity subsides and the organism shows signs of recovery. If, however, the stressor persists, the stage of exhaustion ensues and hyperactivity of the cortex again comes into play (Selye, 1946).

The evidence as presented by Selye (1946, 1949) to support these concepts is clear-cut and generally accepted. The picture becomes obscure about exactly what takes place in the adrenal cortex if the stress continues until the organism dies. There seems to be a question whether the stage of hypersecretion of cortical products continues until death ensues or if the opposite occurs and the cortex fails and the organism succumbs in a state of hypoadrenalism.

Grossly, the adrenal is heavier than normal and the cortex appears hypertrophied in animals and humans dying after prolonged stress (Selye, 1937; Armstrong and Heim, 1938; Sarason, 1943; Tepperman *et al.*, 1943). This is not a positive indication, however, of hyperfunction of the gland at the time of death, since changes occurring previously might have persisted in spite of subsequent failure.

Sarason (1943) and Rich (1944) both describe histological changes in the adrenal glands of patients dying from acute illnesses which they interpret as resulting from an increased demand for adrenocortical hormones. Selye (1949, p. 852) describes similar degenerative changes, but also points to the cellular hypertrophy during the exhaustion stage which "may or may not be accompanied by an overproduction of corticoid substances" (Selye, 1946). The haemorrhagic changes in the adrenal following severe burns and in meningococcal infection (Waterhouse–Friderichsen syndrome) are well known (Hartman and Brownell, 1949).

It is obvious from these conflicting reports that morphological studies are inconclusive. Selye (1950, p. 299) summarizes by stating: "During the stage of exhaustion cytolytic phenomena, infarction and necrosis prevail; otherwise the changes resemble those seen in the alarm reaction (corticoid overproduction). A more detailed characterization is not yet possible, since comparatively few data are available concerning adrenal changes in this stage."

Adrenal functional studies during exhaustion are scarce. Selye (1950, p. 223) states: "Unfortunately, up to now no systematic investigations have been published concerning the urinary elimination of corticoids throughout the three stages of the general adaptation syndrome. Presumably, in the stage of exhaustion the adrenal function becomes deficient when the structure of the gland breaks down."

Selye (1950) noted, however, that in chronically ill and debilitated patients the corticoid secretion (p. 223) as well as the 17-ketosteroid excretion (p. 227) is low. He felt that this corresponded to the stage of resistance when corticoid secretion is less intense than during the alarm reaction. Almy *et al.* (1950), in a similar group, noted a poor eosinopenic response to adrenaline which led him to suspect that inadequate adrenal function may play a part in determining the course of illness. Furthermore, the clinical picture of the terminally ill debilitated patient resembles that of Addison's disease.

Thus the meagre evidence available at the present time suggests that the adrenal cortex breaks down during the exhaustion stage of the general adaptation syndrome, and this failure may be involved in, or even lead to, the death of the organism. On the other hand, the evidence is not unequivocally in favour of this hypothesis, and some of the studies seem to indicate that there is actually adrenal hyperfunction during this stage. A clarification of this enigma would have obvious clinical significance. If the adrenals were proved to be in a state of failure in the terminal stages of illness, treatment with the available cortical hormones would be expected to prolong life, and in some cases might actually prevent death by maintaining the resistance of the organism until the stress could be eliminated.

An accurate method for measuring adrenal cortical activity has been added to the medical literature—the measurement of the circulating eosinophil level (Thorn *et al.*, 1949). This index was used when functional studies were carried out during some phases of the adaptation syndrome. A pronounced eosinopenia

(which indicates adrenal hyperactivity) has been shown to occur during the alarm reaction, with a return to normal levels during the stage of resistance (Dalton and Selye, 1939). Selye (1950, p. 405) states, however, that "no systematic investigations [of haematological changes] have yet been carried through to the later periods of the stages of resistance and exhaustion."

In order to help clarify the functional status of the adrenal cortex during the final phase of exhaustion and at death, a study of the level of circulating eosinophils in a selected group of terminal cases was undertaken.

Methods and Materials

A total of 325 eosinophil counts were done on 116 individuals. These were divided into a control group and an experimental group (Table I). The latter consisted of

TABLE I.—*Summary of Results*

	No. of Patients	No. of Counts	Counts <30/c.mm. No.	Counts <30/c.mm. %
Controls	54	108	11*	10
Experimental group:				
Survivors	12	75	39	52
Dead	50	142	105	74
Total	116	325	155	47·7

* Acutely ill patients.

62 moribund patients with a variety of diseases, and was further subdivided into 12 who survived and 50 who died. All patients were selected from the wards of the Atlantic City Hospital, the criterion for selection being the clinical impression that the patient was critically ill and probably would not survive.

Serial eosinophil counts (daily when feasible) were done up to the time of death or until the patient had recovered sufficiently to be sitting up in a chair. All patients who died had counts within 24 hours of death and as close to the time of death as possible. In five instances blood drawn from the jugular vein within one hour of death was used, since there was an exact correlation between the level of eosinophils immediately before death and the level within the four hours after death. This correlation was confirmed in a small series of patients.

TABLE II.—*Comparison of Eosinophil Counts on Control Groups with Final Counts on Dying Patients*

Group	No. of Patients	No. of Counts	Range Low	Range High	Average Eos./c.mm.	Stand Dev.	% Below 30 Eos./c.mm.	Critical Ratio
Control A	8	8	211	1,931	830	650	0	3·5
,, B	15	15	44	272	149	63	0	8·3
,, C	6	60	5·5*	471	116	84	8*	9·9
,, D	25	25	0*	416	84	79	24*	4·8
Experimental	50	50	0	78	7·2	15	94	—

* Patients acutely ill or receiving A.C.T.H.

$$\text{Critical ratio} = \text{diff. between averages} \div \sqrt{\frac{(\text{stand. dev.})^2_{\text{control}}}{\text{Number (con.)}} + \frac{(\text{stand. dev.})^2_{\text{exp.}}}{\text{Number (exp.)}}}$$

Critical ratio of 3 or above establishes significance. (1,000 to 1 chance of results being reversed on a repetition of the experiment.)

The control group consisted of 54 patients, on whom 108 eosinophil counts were done (Table I). These patients were subdivided into four groups (Table II) as follows:

Group A consisted of eight individuals with a known allergic background and an eosinophilia as shown by peripheral blood smear. These were included merely to assure the reliability of our counting method.

Group B was made up of 15 healthy hospital employees selected at random.

The patients in Group C were selected as follows: six beds were picked at random from the medical ward and daily eosinophil counts were done on the next patient admitted to that bed.

Group D consisted of 25 patients selected at random by the haematology laboratory technician while doing routine blood counts throughout the hospital.

The stain and direct counting technique used was as outlined by Pilot (1950).

Investigations by Rud (1947) in Norway, who did 27,000 eosinophil counts, and Best and Samter (1951) in the United States have shown that there is a wide variation in the normal range of eosinophils (30–250 per c.mm.), with a mean of 126. Even though a count of 30 cells is considered eosinopenic by some authorities (Recant *et al.*, 1950), this was accepted as the lower limit of normal for this study.

The American authors also point out that there are rapid and appreciable short-term fluctuations as well as diurnal variations. In allergy, counts up to 1,500 were common and occasionally rose as high as 4,000 eosinophils per c.mm. However, normal individuals were found to maintain within broad limits the same general level of eosinophils over prolonged periods. The authors therefore conclude that the average change in a sufficiently large, homogeneous, well-controlled group should be a significant measure of pituitary-adrenal function (Best and Samter, 1951).

An attempt was made to determine the eosinophil level as close to the time of death as possible; consequently conditions such as fasting, time of day, etc., could not be controlled. However, counts were done at all times during the night and day and under a variety of conditions, and therefore should represent an unbiased sample.

Results. I: Control Group

A total of 108 eosinophil counts were done on 54 individuals, with the following results (Table I). Of the entire group only 10% had fewer than 30 eosinophils per c.mm.

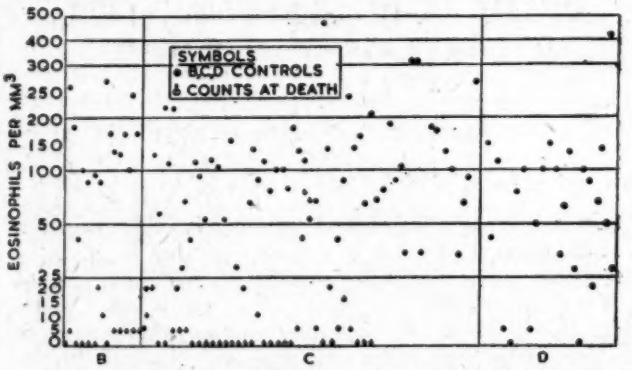

FIG. 1.—Comparison of Results in Control Groups B, C, and D with the results in the experimental group. This graph shows that most of the counts in the control groups are normal, in contrast to the low counts in the patients who died.

Breakdown into subdivisions showed the following (Table II and Fig. I):

Group A: Eosinophilia.—All subjects had a circulating eosinophil level above 200, the maximum being 1,931 per c.mm.

Group B: Healthy Hospital Personnel.—This group averaged 149 eosinophils per c.mm., with a range of 44 to 272.

Group C: Serial Counts on Six Patients Selected at Random.—The average count was 116 for this group, in spite of the fact that they were ill patients undergoing stress. Only five of the 60 counts were below 30, and four of these were on patients with a recent myocardial infarction, which is known to cause an eosinopenia (Recant *et al.*, 1950; Gabrilove, 1950).

Group D: Single Counts on 25 Patients Selected at Random.—This group averaged 84 eosinophils per c.mm., with over three-fourths of the patients having normal counts

in spite of the fact that many were acutely ill. This is actually a very poor group of controls, although (as with Group C) biased in the opposite direction, since it contained patients with the following conditions: ruptured appendix, one-day post-gastrectomy, spontaneous abortion, acute infections, and one patient who was receiving A.C.T.H. The entire 24% of eosinopenic results occurred in these patients, all of whom had conditions known to cause a reduction in eosinophils (Laragh and Almy, 1948; Davis and Hulit, 1949; Coppinger and Goldner, 1950; Gabrilove, 1950; Roche *et al.*, 1950).

The control group taken as a whole tends to correlate with previous studies (Rud, 1947; Best and Samter, 1951), and hence assures the validity of the technique used in this study. Furthermore, this group demonstrates that even in hospital patients, except when very acutely ill, a marked eosinopenia seldom occurs.

II: Moribund Patients

A total of 142 eosinophil counts were done on 50 moribund patients who succumbed to their illness: 94% (47 of 50) had fewer than 30 eosinophils per c.mm. on the day of death. In fact, 56% (28) had none and 82% (41) had a count of less than six. Tests of reliability were applied, the counts being compared at death with each of the control groups, and the differences were found to be statistically significant (Table II).

The three patients with counts above 30 were unique in that they all died suddenly though previously well. All three had been carrying on normal activity until less than five hours before death. It is felt that the eosinophil level had not had enough time to respond fully to the stress. It is probable that if they had lived a few more hours they too would have had an eosinopenia.

It is known that the normal eosinopenic response to adrenal cortical activity is a delayed one (Last *et al.*, 1950) with a gradual decline to subnormal levels over a period of hours (Forsham *et al.*, 1948). In fact, this decline is usually preceded by a transient eosinophilia (Forsham *et al.*, 1948).

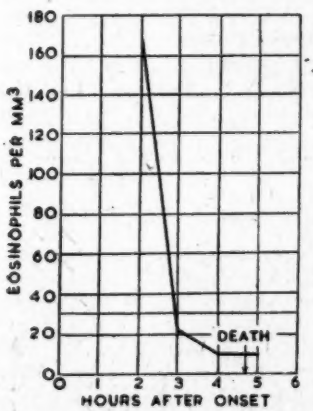

FIG. 2.—Falling eosinophil level in a dying patient. This patient died in a few hours from a cerebral haemorrhage. This is an accelerated example of the typical eosinopenic response found in the patients who died. The two-hour level may represent the end of the initial eosinophilia usually demonstrated immediately after onset of stress.

Fig. 2 represents an accelerated example of the typical eosinopenic response found in the dying patients. The eosinophil count in this woman was at a normal level two hours after the onset of a cerebral haemorrhage, but fell rapidly to a low level before her death. Recant *et al.* (1950) describe a similar response in a patient with an acute coronary occlusion.

The general trend in the experimental group was a slowly falling level of eosinophils up to the very moment of death. If the patient remained moribund over a protracted period of time the eosinopenia persisted without fluctuation. It often remained at the zero level for days. Of the 142 counts done on this group, only 37 were above 30 eosinophils per c.mm.

The diseases responsible for death in these patients constitute a representative sample of those usually seen in an active general hospital. Cerebrovascular accidents, malignancy, and heart disease head the list. The majority of the patients were in the older age groups.

The average length of stay in hospital was 7.4 days, and the range was from "dead on arrival" to 96 days. However, 34% died within 24 hours of admission and 74% within the first week.

III: Survivor Group

These 12 patients were moribund, and were considered to be terminal cases when serial eosinophil counts were begun. The patients recovered, and, since the results proved interesting, they are reported. This group serves as a check on both the experimental and the control groups, since the initial counts were low, as in the former group, and the subsequent counts normal, as in the latter group.

All these patients had an initial eosinopenia—in fact seven (58.3%) had no eosinophils. All counts had risen to normal levels by the time the patients had recovered enough to be up in a wheel-chair.

These results parallel those of Elliott (1950), working with patients in congestive failure, and also with Gabrilove's (1950) group of patients with coronary occlusion and those who had had an operation. Hain (1951) described similar changes in children with acute febrile illnesses.

Discussion

This is the first attempt to study the function of the adrenal gland in terminal states by observations on its physiological activity, although Recant *et al.* (1950) noted eosinopenia in several patients in their group who were in terminal stages of disease.

It is apparent from the results that, if the circulating eosinophil level is a reliable index of corticoid activity, there is no failure of the adrenal cortex during the terminal stages of illness. In fact, the evidence presented here indicates that the cortex is in a constant state of hyperfunction and hypersecretion until the very moment of death. If this be true, supplementary therapy with cortical hormones would be of no value in the moribund patient unless specifically indicated by the nature of the disease.

The findings also suggest that the return to adrenal hyperfunction demonstrable as an organism passes from the stage of resistance of the general adaptation syndrome to the stage of exhaustion persists throughout its entirety.

Dr. S. Sevitt (1951), studying the eosinophil level in burned patients, reached a similar conclusion to my preliminary report on eosinophil levels at death (Jennings, 1951). He states: "Therefore the eosinophil changes in five out of eight fatal cases suggested that hyperactivity of the adrenal cortex was present before death: in another patient there was evidence suggesting that adrenocortical failure had occurred, and in two others the evidence was inconclusive."

Since the life of the eosinophil is believed to be of short duration it might be postulated that failure of the bone marrow to continue production of these cells may occur terminally and account for the eosinopenia. This seems unlikely in the light of all the investigations on stress and the speed with which the eosinopenia can take place (Fig. 2). This would also be inconsistent with the rising leucocyte count which often occurs as a terminal event. Such a theory could readily be disproved by demonstrating an eosinophilia in a terminal untreated case of Addison's disease.

There is a lack of correlation between the findings reported here and the well-known acute adrenal insufficiency demonstrated by the Waterhouse–Friderichsen syndrome and following severe burns (Hartman and Brownell, 1949). In both of these conditions there is apparently primary haemorrhage into the adrenals. Nevertheless, Dr. Sevitt's observations on fatally burned patients indicated adrenal hyperfunction, so histological findings may not be a reliable index of cortical function.

Our studies shed no light on the fate and function of the eosinophil during stress, but our results in acutely ill patients seemed to parallel the work of previous authors who demonstrated a fall in the eosinophil count during the acute stress (Elliott, 1950; Gabrilove, 1950; Hain, 1951) and a return to normal during recovery.

Nevertheless, the eosinophil level seemed to be of only slight prognostic significance, since it was low in all acutely ill patients even when it was apparent from the nature of their illness that they were not critically ill. The rise in eosinophils usually lagged behind the appearance of clinical improvement, similar to the delayed fall seen in stress.

However, a rising level of eosinophils in a patient who continues to appear moribund is a good prognostic sign. This was recognized by clinicians years ago (Sodeman, 1950).

In rare instances a very low level may be an indication of impending death in a patient in whom this is not apparent clinically. This occurred in two of our patients in whom an eosinopenia was discovered before the approach of death was apparent.

This study is limited, but is presented in order to stimulate further exploration into the changes which take place in the organism before death. A larger group of terminal patients should be investigated in an attempt to correlate the histological appearance of the adrenals with the level of corticoid secretion and the eosinophil level as well as any other adrenal functional studies which may be available. This should yield valuable information about the hormonal changes occurring at the end of the exhaustion stage of the general adaptation syndrome.

Summary

A carefully controlled series of 50 moribund patients suffering from a variety of diseases was followed by serial eosinophil counts until death. In 47 (94%) the eosinophil count fell to zero or close to zero and remained so until death. Suddenness of death seemed to be an important factor in the three patients who failed to develop an eosinopenia. The eosinophil count rose from low levels to normal in 12 other patients who recovered from the moribund state.

This study indicates that the adrenal cortex apparently does not fail during prolonged stress, but continues to show hyperactivity up to the very moment of death; consequently supportive therapy with cortical substances is not indicated in terminal states.

The eosinophil count seems to be of only limited prognostic significance in such cases, since changes usually accompany or lag behind the clinical findings rather than precede them.

I am grateful to Dr. Milton Ackerman, pathologist at the Atlantic City Hospital, for his consistent co-operation and help during the course of this work. I am indebted to Miss Evelyn Kress, who did most of the eosinophil counts, and to Miss Clara Louise Kuehn, who prepared the manuscript. I also wish to express my sincere thanks to Dr. Hans Selye, Director of the Institute of Experimental Medicine, University of Montreal, Canada, for reading this paper and offering many helpful suggestions.

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THE CONDUCT OF PREGNANCY COMPLICATED BY DIABETES MELLITUS

BY

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Most authorities advise and the majority of maternity centres practise early termination of pregnancy at about the 36th week in diabetic women. It is well recognized that intrauterine death may occur in the later weeks of pregnancy, and the aim of early termination is to remove the foetus from the harmful influence of the diabetic expectant mother. Recent work (Fouracre Barns *et al.*, 1950) suggests that this harmful influence may be due to overactivity of the maternal anterior pituitary gland. If this is so, the foetus will have been subjected to it throughout pregnancy, and intrauterine death may represent a late effect. However, early termination of pregnancy, whilst removing the foetus from the influence of the lethal factor, produces an immature infant who has already been subjected to its harmful effects and who is less able to withstand extrauterine life than an immature infant of a normal mother.

Early Termination

Peel and Oakley (1950) recommended early termination of pregnancy. In their series of 123 diabetic pregnancies there were 12 in which intrauterine death occurred (presumably before induction of labour or caesarean section was due to be carried out). In the remaining 111 treated by premature termination of pregnancy four babies were fresh stillborn and 20 died in the neonatal period. Similar results with early termination of pregnancy were reported by White (1949) in a series of 361 diabetic pregnancies. Neonatal death is thus a large factor in the production of the total foetal death rate in diabetic pregnancies treated by early termination. Since in most clinics early termination is advised, it is not surprising that there are few recorded individual series of diabetic pregnancies which have been allowed to proceed to spontaneous termination.

In a previous article (Fouracre Barns, 1950) an attempt was made to evaluate the results of termination against those when the pregnancy was allowed to proceed to spontaneous termination. The foetal salvage rate in 22 diabetic pregnancies terminated at 36 weeks was 41%, whereas in 23 diabetic pregnancies treated over the same period and allowed to terminate spontaneously the salvage rate was 43.5%. Thus the foetal salvage rate for the two methods of delivery was approximately the same. Although intrauterine death occurred more often when the pregnancy was allowed to proceed (43.5% as compared with 27.3%), neo-

Case No.	Age (Years)	Duration of Diabetes at Time of Pregnancy (Years)	Previous Obstetric History					Present Pregnancy					Remarks
			Date	Delivery	Duration (Wks)	Result	Wt. of Baby	Duration (Weeks)	Delivery	Wt. of Infant	Result	Feeding	
							lb. oz.			lb. oz.			
1	27	1		Nil				37 1/7	Forceps	7 11½	A	Breast+suppl. →art. feed	
2	37	15		,,				37 1/7	,,	7 7	A	Breast+suppl. →art. feed	Benign hypertension
3	26	18		,,				38 3/7	C.S.	6 0	A	Breast+suppl. →art. feed	C.S. for uterine inertia. Puerp. pyrexia—urinary infection
4	25	9		,,				35 3/7	Spont.	8 1	A	Breast+suppl. →art. feed	Puerp. pyrexia—pleurisy, hypertension
5	27	8		,,				38 4/7	,,	7 11½	A	Breast	Mild hypertension
6	30	10		,,				41	C.S.	9 8	A	Breast+suppl. →art. feed	Hydramnios+C.S. for large baby and post-maturity
7	42	12	*1925	Spont.	40	A	9 8	38 3/7	Forceps	8 13	A	Breast+suppl. →art. feed	Hypertension
			*1927	,,	40	A	9 0						
			*1930	,,	40	A	9 8						
			*1931	,,	40	A	9 4						
			*1934	,,	40	A	11 8						
8	34	6	*1935	,,	40	A	5 12	38 2/7	Spont.	5 12	A	Breast	Toxaemia
9	28	2	*1942	Spont. breech	40	A	7 0	38 3/7	,,	9 6	ND	—	Venous thrombosis at 37 weeks—treated with anti-coagulants Baby died after ½ hr. c punctate haem. of brain
			*1945	Spont.	40	A	6 9						
10	22	7		Nil				38 6/7	Forceps	8 13	A	Breast →art. feed	
11	28	2	See Case 1					38 4/7	,,	9 14	A	Breast →art. feed	
12	29	7		Nil				37 1/7	S.I.	3 8½	M	—	Intrauterine death at 33 6/7 week
13	36	4	*1932	Spont.	40	A	8 12	38 5/7	Spont.	10 11	A	Breast+suppl. →art. feed	Hypertension
			*1934	,,	40	A	8 6						
			*1936	,,	40	A	8 14						
			*1940	,,	40	A	9 0						
14	40	18	1937	,,	40	M	3 7	38 1/7	,,	6 6½	M	—	Intrauterine death at 37 4/7 week
			1938	C.S.	36	A	5 14						
			1941	,,	36	ND	?						
			1942	Spont.	40	M	6 9						
			1944	C.S.	36	ND	6 0						
15	29	7	See Case 12					40	,,	8 13½	A	Breast →art. feed	Hypertension
16	26	1		Nil				40 1/7	C.S.	9 15½	A	Breast	C.S. for severe toxaemia
17	30	4	See Case 9					37 6/7	Spont.	7 4½	A	Breast →art. feed	Hypertension
18	36	10		Nil				35 5/7	Forceps	8 13½	M	—	Intrauterine death at 35 3/7 Hydramnios+
19	22	1						36	C.S.	9 0	A	Breast	C.S. for severe toxaemia
20	37	17	1941	C.S.	36	ND	7 7½	40 1/7	,,	11 6	A	Breast+suppl. →breast	Hydramnios+C.S. for large baby and previous C.S.
			1942	,,	37	ND	7 11						
			1944	,,	37	ND	7 0						
21	28	11	1946	S.I.	36 6/7	SB	6 1	39 3/7	Forceps	8 8	A	Breast+suppl. →art. feed	Hydramnios+
22	27	2		Nil				38 3/7	Spont.	9 13½	A	Breast+suppl. →art. feed	
23	25	10	See Case 10					37	,,	7 15	A	Breast+suppl. →art. feed	Toxaemia
24	27	6	1949	S.I.	36	A	7 8	37 6/7	,,	8 11	A	Breast+suppl. →breast	Hydramnios

C.S.=Caesarean section. M=Macerated. ND=Neonatal death. S.I.=Surgical induction of labour. A=Alive. SB=Stillborn.
*=Prediabetic pregnancy.

natal death was less frequently seen (16.6% as compared with 43.8%). Although these numbers are small, they suggest that early termination does not alter the outcome of pregnancy, but results in neonatal death of the infant rather than in a stillborn foetus.

As a result of these findings it was decided to allow pregnant diabetic women to proceed to spontaneous termination or delivery at term, except where some complication arose necessitating termination for the sake of the mother. Twenty-four diabetic pregnancies treated in this manner at University College Hospital since 1947 are recorded (see Table).

Present Series

We have admitted our patients at the 35th week of pregnancy in order that their obstetric and diabetic states may be carefully observed. In practice it has been found that the diabetes is easily controllable up to term, and in fact the insulin requirement often falls markedly a day or two before delivery (Fouracre Barns and Morgans, 1949). No difficulties have arisen with the diabetes when caesarean section has been necessary. However, during some long and difficult labours associated with the large baby diabetic control has been difficult to maintain, and very careful observation of these patients has been necessary. The majority of the patients received hormone therapy in the form of stilboestrol. This was given in tablet form by mouth from the time of the patient's first attendance, and the daily dose was gradually increased so that she received 160 mg. a day at the 36th week of pregnancy.

In only one patient was it necessary to terminate the pregnancy prematurely. This was done at the 36th week by caesarean section on account of severe toxaemia (see Case 19). There have been one neonatal death and three intrauterine deaths, the details of which are given in the table. Two of the mothers of these four unsuccessful pregnancies each had a subsequent pregnancy with the birth of a live infant (Cases 15 and 17). It will be noted that two of the three intrauterine deaths occurred before the 36th week of pregnancy, the usual time for inducing labour in diabetic pregnancies.

Two other patients were of special interest. One patient (Case 20) had three caesarean sections at the 36th to 37th week of pregnancy prior to 1947. On each occasion the baby lived a few hours only. Recently she was allowed to proceed to term, and caesarean section was carried out one day after the expected date of delivery on account of the large baby and the previous caesarean sections. She was delivered of a live baby weighing 11 lb. 6 oz. (5.1 kg.), which survived. In 1946 the other mother (Case 21) had a stillborn infant following a pregnancy terminated at the 36th week. Death was due to cerebral haemorrhage. Recently she was allowed to proceed to spontaneous termination, which occurred at the 39th week of pregnancy, and she was delivered by forceps of a live baby.

It will be seen from the table that a large proportion of our mothers went into spontaneous premature labour at the 37th to 39th week. It seems possible, therefore, that the diabetic mother herself cuts short the influence of the lethal

factor in such cases and yet allows the infant the longest possible time *in utero* compatible with intrauterine survival.

Cases 15, 17, 20, and 21 had had a previous diabetic pregnancy which resulted in a dead infant. The cause of death on two occasions was cerebral haemorrhage (Cases 17 and 21), but in Cases 15 and 20 the diabetic lethal factor was responsible for the foetal deaths. It follows, therefore, that a diabetic pregnancy which results in the death of the foetus need not be followed by subsequent unsuccessful diabetic pregnancies.

It is well recognized that the diabetic mother has difficulty in breast-feeding her infant. The cause of this is obscure. There is, however, one factor which tends to hinder her. It is that the immature baby does not suck as well as the full-time baby, and hence the earlier the termination of pregnancy the more likely is it that breast-feeding will not be established. One of our patients (Case 24) illustrates this point. In 1949, at another hospital, she was delivered prematurely at the 36th week, by induction of labour, of a baby weighing 7 lb. 8 oz. (3.4 kg.). The baby was difficult to rear in the early weeks and she was unable to breast-feed it after three weeks' energetic trial. Recently she was delivered spontaneously at the end of the 37th week of a baby weighing 8 lb. 11 oz. (3.9 kg.). This baby was at the breast within 48 hours, and breast-feeding was well established during the second week of the puerperium.

The danger of neonatal death in immature infants of diabetic mothers is real. Premature induction of labour increases this danger and produces an infant which is difficult to rear. On the other hand, by allowing the pregnancy to proceed the possibility of difficult labour and disproportion becomes greater as term is approached. In the present series forceps were needed for delivery on seven occasions and caesarean section was performed twice on account of a large infant. The obstetrician's task, therefore, of producing the less immature live infant is increased while that of the paediatrician is decreased.

Conclusion

The foetal salvage rate in this small series (20 out of 24) would appear to justify the course taken of not prematurely terminating the pregnancy. Our improved results are possibly due to the hormone therapy which these mothers have received. It is, however, more likely to be due to the close co-operation between physician, paediatrician, and obstetrician in the care of the diabetic mothers and their offspring.

We should like to record our gratitude to Professor W. C. W. Nixon, director of the obstetric unit, for permission to report these cases, and to the members of the paediatric unit under the direction of Dr. B. E. Schlesinger for their invaluable help in the care of the infants.

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In accordance with an international agreement the Government of the United Kingdom can insist on the international recognition of yellow fever inoculations performed in this country only if: (*a*) they are done at a centre or by a medical practitioner specifically authorized by them for that purpose; (*b*) the certificate is given on one of the international forms specially supplied to such centre or practitioner; (*c*) a vaccine approved by W.H.O. is used for the inoculation. Certificates issued other than in accordance with these conditions will probably not be accepted by foreign health authorities. A list of places where yellow fever inoculation is given free of charge will be found on the back of the notice to travellers issued with all renewals of passports or can be obtained from travel agencies or the Ministry of Health.

TWO CASES OF DIABETES MELLITUS IN INFANTS UNDER 1 YEAR

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Although the onset of diabetes mellitus occurs in 5 to 6% of cases in the first ten years of life, it is rare for the disease to develop in infancy. John (1946) listed 85 cases of diabetes in infants under 1 year. Schwartzman, Crusius, and Beirne (1947), in a more detailed review of the world literature, summarized 57 cases under 1 year and gave details of most of them. The first to survive in order of time was the 27th they describe, and was reported in 1925. Since then only 11 have survived out of the 24 of which adequate details are given. It is not easy to judge how many of these cases had passed into diabetic coma.

In the following two cases which survived, the symptoms began when the patients were under 1 year old.

Case 1

A baby boy aged 13½ months was admitted to hospital on June 18, 1950, with severe glycosuria and ketosis. His brother, aged 10, regularly attended the diabetic clinic, and their parents showed great intelligence and skill in keeping his diabetes stable. Neither of them was diabetic, and there was no known diabetes in either of their families.

The baby weighed 7¼ lb. (3.3 kg.) at birth on May 3, 1949. He did well, weighed 22 lb. (10 kg.) in December, 1949, and was weaned by February, 1950. A few hours after receiving a second dose of antidiphtheria vaccine on March 19 he lost his appetite. He began to vomit solid food and became very constipated. This continued, and by the time of admission he had lost 8 lb. (3.6 kg.). By the middle of May he had become very thirsty, and developed polyuria, and by early June he had become so feeble that he fell over if sat upright. Just before admission his parents tested his urine, and found a full reduction of Benedict's solution.

On admission he had a good colour, but was drowsy and very fretful. The eye tension was lowered, the tongue dry, and the skin dry and inelastic. He was teething, but there was no evidence of infection. His temperature was 96° F. (35.6° C.), pulse 108, and respirations 24. The urine gave full reduction of Benedict's solution and strongly positive Rothera and Gerhardt reactions. The blood sugar was 440 mg. per 100 ml.

He was given either 10 or 15 units of soluble insulin subcutaneously at six-hourly intervals, and at 10.30 a.m. next day the blood sugar was 90 mg. and the ketosis was receding. The next insulin was given 11 hours later, when the blood sugar had risen to 540 mg. per 100 ml. After a short time on six-hourly insulin again it was found that eight-hourly doses (8 a.m., 4 p.m., and 12 midnight) controlled him adequately. The night dose was only two units, but this made all the difference between a blue and orange early-morning urine test. He was not weighed on admission, but was 22¼ lb. (10.3 kg.) 10 days later. He was given 130 g. carbohydrate diet, which he took well. A variety of combinations of insulin were tried in order to eliminate three

injections, and during this time he developed bronchitis and laryngitis and later diarrhoea. These infections disturbed the diabetes to some extent, though he never developed any ketosis. Difficulty was experienced in getting urine specimens, and it was often necessary to wring out his napkins for a test. He was discharged, after nearly three months, on 18 units of globin insulin once daily; he then weighed 27 lb. (12.2 kg.).

He attended the diabetic clinic for the next six months and remained well, gaining weight rapidly. He was kept on globin insulin, though occasionally an additional 2 units of soluble insulin was given at 11 p.m. if a specimen at that time was loaded with sugar.

In March, 1951, he was readmitted with a blood sugar of 300 mg. per 100 ml. and a cough and catarrhal otitis media, which failed to respond to penicillin, but responded to sulphadimidine. The dose of globin insulin had to be increased to 34 units, but this did not control him satisfactorily. He was discharged on 18 units of soluble insulin at 8 a.m. and 12 units at 4 p.m. and 90 g. carbohydrate diet, as he had gained 11 lb. (5 kg.) in six months. On October 25 he weighed 45 lb. (20.4 kg.), was on 100 g. carbohydrate diet, and was having 12 units of soluble insulin at 8 a.m. and 13 units at 4 p.m.

Case 2

A female infant aged 11 months was admitted to hospital on March 1, 1951, at 3 p.m. She was an only child who had had no previous illnesses and was quite well until the day before admission. There was no family history of diabetes, and glycosuria was absent in the parents. On February 28 the child vomited once and was said to look very ill. She was putting her hand to her head; she had previously done this when cutting teeth. Her parents thought she seemed better on March 1, but she was not taking notice of her surroundings. She was sent into hospital by her doctor.

On admission the child looked moribund, and was a bluish-grey colour, with sighing respirations and active alae nasi. A note was made, however, that the clinical picture was not typical of lobar pneumonia, although she had an impaired percussion note at the right base and moist rales at both bases, especially on the right. The throat was clear and the eardrums were normal. There was a black necrotic area of skin on the buttocks. Clinically, there was a moderate degree of dehydration. A definite left-sided facial weakness was noted and ? slight ptosis of the left eye. Deep reflexes were normal, pupils reacted to light, and the fontanelle was not tense. There was no neck stiffness and Kernig's sign was negative. Temperature, 98° F. (36.7° C.).

The diagnosis was in doubt, but as the child was moribund 500,000 units of crystalline penicillin and 1 g. of sulphadimidine were given intramuscularly at once, and the child was placed in an oxygen tent. Lumbar puncture showed a crystal-clear fluid, not under increased pressure, with no increase of cells. A catheter specimen of urine showed a distinct cloud of albumin, with full reduction with Benedict's solution, and the deposit contained some casts and a few epithelial cells.

In view of the large amount of sugar in the urine we wondered if we were dealing with a case of diabetic coma with severe air hunger. Intravenous blood was taken at 8 p.m. for sugar estimation, but before the result was known an intravenous drip of half-strength saline with 5% glucose was set up and 10 units of soluble insulin was injected into the drip. Three minutes later the child opened her eyes a few times, moved her head and limbs, and appeared to be waking. Ten minutes later 10 units of soluble insulin was given intravenously. The result of the blood-sugar estimation had then become known, and was 1,160 mg. per 100 ml.

Her immediate further progress is best summarized as follows. *11 p.m.:* Blood sugar 1,080 mg. per 100 ml. A total of 50 units of soluble insulin had by then been given. Colour improved and respirations less deep. *March 2, 1 a.m.:* Urine still deep orange with Benedict's solution. Child a good colour, respirations quiet, tongue moist, and child moving freely. 35 oz. (1 L) of fluid intravenously had been given in previous five hours. *2 a.m.:* Urine still deep orange with Benedict's solution. *3 a.m.:* 20 units of soluble insulin given subcutaneously. *5.30 a.m.:* Blood sugar 500 mg. per 100 ml. *9.30 a.m.:* Blood sugar 300 mg. per 100 ml.; blood chlorides 650 mg. per 100 ml. *2.30 p.m.:* Blood sugar 70 mg. The child then had symptoms of hypoglycaemia, which were corrected.

From then on the child improved and the insulin dosage required was worked out. She was eventually discharged well on June 8 on a diet of 1,435 calories (carbohydrate 135 g., protein 86 g., fat 55 g.). Her insulin requirements were 9 units of soluble at 7.30 a.m. and 10 units at 4 p.m. Her weight shortly after admission to hospital (March 10) was 18 lb. (8.2 kg.), and on discharge it was 22 lb. 4 oz. (10.1 kg.).

She has since been seen at the out-patient department on several occasions, and is progressing well. Owing to her rapid gain in weight the carbohydrate in her diet has been reduced to 120 g. and the insulin to 8 units at 7.15 a.m. and 9 units at 4.30 p.m. Her weight on October 18 was 25 lb. (11.3 kg.). A catheter specimen of urine showed no abnormality.

Comments

The highest blood-sugar levels recorded in the review by Schwartzman, Crusius, and Beirne (1947) were 600 mg. per 100 ml. in an infant who survived, and 952 mg. per 100 ml. in one who died.

Case 2 had a blood sugar of 1,160 mg. per 100 ml. before treatment began, the highest figure which we can find recorded in an infant under 1 year.

Newcomb and Farrell (1951), in a review of six cases under 1 year, found fever and pulmonary symptoms in 100%, and "rales and signs of pneumonia" in 66%. They point out that the prevalence of pulmonary signs and symptoms has not been stressed enough. Case 2 fitted into this picture.

We came to the conclusion that soluble insulin at 8 a.m. and 4 p.m. controlled the babies most effectively. Globin insulin alone once a day was satisfactory in Case 1, until the dose rose to over 30 units. One of us (H. S. B.) finds globin insulin once a day most effective throughout the 24 hours in adults, provided the dose remains under 40 units a day. If this amount is exceeded some other combination of insulin is necessary, and it seems that this critical level is lower in infants.

Both babies, when stabilized, showed a marked tendency to gain weight excessively on 130 g. carbohydrate diet. It was found necessary to reduce it after an initial period.

Summary

Two further cases of diabetes mellitus, which began in infants under 1 year old, are described.

The first had a gradual onset, which followed immunization, and was comparatively mild. The second was acute and severe, presenting pulmonary symptoms and signs; her blood sugar was 1,160 mg. per 100 ml., the highest recorded under the age of 1 year.

Both patients survived, and continued well on two doses of soluble insulin daily.

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CARE OF THE NEONATE: INSTRUCTION OF THE MEDICAL UNDERGRADUATE

BY

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The subject of "methods in medical teaching" was considered from many angles at a conference arranged by the Postgraduate Medical School (*British Medical Journal*, 1951, **1**, 1505). Special problems arise in connexion with the instruction of the undergraduate in the care and management of the newly born infant. Thus the hours during which a student has access to the lying-in wards are limited. The wards and nurseries may be closed at just those times when he is free. Control over visits to maternity nurseries is necessary in the babies' interests. The larger the number of students the more difficult it is to meet the needs of teaching instruction without exposing the nursery communities to unjustifiable risk. Furthermore, the readiness with which the student gets access to the newly born babies is considerably influenced by the attitude of the ward sisters. Much, too, depends upon the keenness of the individual student. Always there is the basic problem that in hospital the student studies care of the neonate under conditions which differ widely from those in general practice.

There is no one ideal method for instruction of the medical student in neonatal care. The methods adopted vary from teaching centre to teaching centre; this is particularly true if account be taken of the instruction given in medical schools abroad. This article aims at describing the methods of teaching which have been gradually evolved at the Leeds Maternity Hospital.

Clinical Facilities and their Background

In all, 77 lying-in beds are accommodated in four lying-in wards. Each lying-in ward has a nursery, which serves also as an infant toilet and changing-room. Babies retained in the nursery are limited to those who are excessively noisy and those who require special nursing or medical supervision. Otherwise infants remain with their mothers in the lying-in ward throughout the 24 hours.

A resident paediatric registrar is responsible to the visiting children's physician for the immediate clinical supervision of healthy and ill infants, and receives help in the routine examination of babies from a medical officer of the City of Leeds Maternity and Child Welfare Department, acting in the capacity of clinical assistant. The registrar works in the closest co-operation with the labour- and lying-in-ward sisters. In practice the paediatric registrar is present at almost all premature, difficult, and operative deliveries. Senior members on the whole-time staff of the university department of paediatrics and child health are actively associated with the work among babies in the hospital, undertake a considerable amount of the teaching of students, and are responsible for pursuing agreed lines of research.

The Students

The students for whom instruction in the neonate is arranged are those who are taking out their three months course in obstetrics. Most are in their sixth and final year of study, the remainder being in their fifth year. Accommodation arrangements resemble those at other teaching centres. Throughout the three months of their obstetrical course the students reside in a university hostel reserved for their use and situated immediately adjacent to the Maternity Hospital. The limits of hostel accommodation ensure that the number of students in any one three-month period does not exceed 17. Deliveries are undertaken in hospital and on the district. Midwives with whom the student works in hospital are on the staff of the teaching hospital board, whereas those with whom he works on district are on the staff of the municipal public health department.

At the beginning of each three-months course the newly arrived students nominate a representative who is made responsible for drawing up rosters and with whom the paediatric registrar arranges for instruction to take place at times which do not conflict with ward requirements or with students' other commitments.

Methods of Instruction

The methods of teaching employed may be summarized as follows.

1. *Introduction.*—The purpose of the present teaching is to give the student a sound theoretical and practical knowledge which, by being general rather than specialized, will enable him to deal effectively and confidently with the management of the neonate, under conditions of general practice and in collaboration with his nursing colleagues. This objective is explained to the student at the outset. He is encouraged to study for himself the contrasting circumstances of district care of the newly born on the one hand and of hospital care on the other. He is forewarned against seeing care of the child as something apart from care of the mother. It is explained that the function of paediatric care in hospital is to assist and not to encroach upon the obstetrician's professional responsibility to the mother for the condition of her newly born baby; and the student is reminded that when he enters general practice his responsibilities will extend not only to mother and child but to the entire family. Instruction is integrated with and does not overlap that given by members of the obstetrical staff. At the same time the student is urged to consider his study of the neonate in relation to the teaching he receives elsewhere on the domiciliary, welfare, and hospital care of older infants and children.

2. *In the Labour Ward.*—The number of students present at a delivery may vary from one or two to a dozen, depending upon the time of day at which it takes place. Again, depending upon the circumstances of the delivery, the toilet of the infant immediately after birth and the resuscitative measures are explained by the visiting obstetrician conducting the case, the paediatric registrar or the labour-ward sister. If the paediatric registrar has to be called from other duties to examine a baby the labour-ward sister explains to the students the features of the child's condition which have necessitated the calling in of what may be called "medical aid."

3. *In the Nurseries and Wards.*—(*a*) Each student accompanies the paediatric registrar on organized visits to babies in the nurseries and wards on at least three occasions. Additional nursery rounds are made with the visiting physician. Not more than three students take part in any one visit. Under supervision the student examines babies chosen to illustrate health and disease in both full-term and premature infants. Babies with evidences of infection, trauma, or congenital anomalies are discussed from the point of view of diagnosis, treatment, and prognosis. Special attention is paid to discussion of the general progress and the feeding management of those infants previously delivered by the students taking part in the nursery round. In the event of a baby's progress or condition requiring to be discussed with the father or mother, and in the absence of contraindications, the student is encouraged to be present. If a visit by a health visitor coincides with a nursery round the opportunity is taken to explain to the student the purpose and value of such visits in obviating difficulties apt to occur after the mother and baby have returned home from hospital.

(*b*) It is the practice of lying-in-ward sisters to carry out a daily "feeding management round." Each student is present on at least three such rounds, successive rounds taking place in different wards. Only one student accompanies the

sister on these rounds, which take place at times when mothers are feeding their babies. The sister summarizes progress in each case, demonstrating the presence of both normal and abnormal features and explaining adjustments in management and feeding occasioned by circumstances related to the mother or infant. Attention is drawn to difficulties attributable to inadequate antenatal preparation. The subjects dealt with in the course of these rounds are reviewed at regular intervals by the sisters and paediatric staff.

(*c*) Acute emergencies are relatively infrequent. When they occur the students are notified, and are thus able to take part in discussions concerning diagnosis and immediate treatment. The arrangements give the student an opportunity of studying the early clinical manifestations of such conditions as haemorrhagic disease, haemolytic disease, or acute intestinal obstruction before they have been modified by treatment. The admission of a premature infant from the district is regarded as an emergency in so far as the student sees the special precautions taken by the midwife who accompanies the baby and has impressed upon him the need for co-operation between the family doctor and the local health authority on the one hand and the hospital services on the other.

4. *On District.*—Students are encouraged to continue for a period of two or three weeks visits, at suitably spaced intervals, to the home of babies whom they have delivered on district, and to utilize the visits for studying the inclinations and reactions of the mother to breast-feeding, and the influence of factors peculiar to the home upon the mother's management and care of her infant. In some of the homes there are home helps. In others help is forthcoming from relatives or neighbours. Occasionally the student is able to visit in company with the midwife, but it has proved impracticable to arrange routine joint follow-up visits. Each student is asked to make brief written comments on the progress and circumstances of one of the babies he has followed up in this way. The results have been altogether encouraging, and the comments which have been received afford evidence of the healthily critical attitude of the student and of his ability to adapt his hospital instruction to the conditions of domiciliary care.

5. *Weekly tutorials* are held. At these, instruction is given on the principles and theories which provide the background to neonatal care. The tutorials complement the essentially individual tuition given during visits to the nurseries and wards. The sister tutor or one of the ward sisters assists at some of the tutorials by explaining and demonstrating practical aspects of nursing care and management of the neonate.

6. Attendances at *necropsies* on infants are not encouraged, but post-mortem results are made known to and discussed with the students.

7. *Discussions.*—At regular intervals senior members of the paediatric staff meet the students as a group to discuss a previously selected subject. Free discussion is encouraged, and, provided it follows profitable lines, strict adherence to the original subject is not attempted. Thus an intended discussion on breast-feeding may develop into one on prematurity. Spontaneous variations of this kind usually result from questions raised by the students themselves. A point of interest has been the frequency with which these questions have been stimulated not by didactic teaching but by observations made by the student himself in the course of his visits to the labour ward or to nurseries or homes on the district.

Together, tutorials and discussions occupy a total of some 20 hours in each three-months period. At the last meeting with each group of students suggestions for improvement of the course are invited. The response has always been constructive and on occasion salutary. Thus, for example, at a time when the teaching methods being employed were still in a fluid state and when a disproportionate amount of time was being devoted to morbidity, a request was made by the students that there should be a shift of emphasis from study of disease to that of health in the neonate. The instruction now given embodies many of the suggestions which have been forthcoming from time to time from the students.

For a period students attended the follow-up clinic which has for many years formed part of the service provided by the hospital. The attendances of students was discontinued for three reasons. In the first place, it was felt that the students' time would be more profitably spent in the hospital or on the district. Secondly, the circumstances under which infants attend the clinic are apt to leave the student with a confused impression. Most infants are already under the care of the family doctor and the welfare clinic. Some are brought because their mothers are attending a post-natal session. Only a small proportion came for organized follow-up on account of prematurity or past neonatal illness. In the third place, the student is given individual instruction at one of the City of Leeds infant welfare clinics as part of his general paediatric course.

Conclusion

The response of the students suggests that the methods of teaching on the neonate are achieving some of their objectives. It is to be emphasized that the methods outlined could not have been organized without the co-operation of professional colleagues in a number of spheres. First attempts and subsequent developments were made possible only by the encouragement and facilities received at the hands of the professor of obstetrics and the senior obstetrical staff of the Maternity Hospital. The matron of the hospital has encouraged senior members of her staff to contribute to the instruction of medical students. The hospital sisters, for their part, have shown a stimulating enthusiasm, doing everything possible to give the maximum assistance and being responsible for many constructive suggestions. Valuable help has been received from the medical officer of health and the supervisor of midwives of the City of Leeds, who have encouraged district midwives to explain to students, wherever possible, the domiciliary management of the neonate. Acknowledgments would not be complete without tribute being paid to the invaluable work of a succession of paediatric registrars, including Dr. P. Rivett, Dr. M. F. Buchanan, Dr. G. Kitching, and Dr. D. Haiste.

What are the advantages to the student of the organized teaching which has thus been made possible? He sees care of the infant as part of care of the mother, and vice versa. The value of antenatal care as a factor in promoting health in infancy is manifest. He gets a practical insight into the physiological and other events which may discourage a mother in her early endeavours to feed her infant at the breast. The intangible no less than the tangible relationships shared by mother and child are more easily appreciated. The student sees at one and the same time health and disease in infants of the same age period, and he appreciates the complementary nature of preventive and curative care. The functions of doctor, midwife, health visitor, and home help are seen in relation to one another. Care of the newly born infant, whether in hospital or in the home, is considered against a background of the family circle.

Local experience has amply confirmed the view that it is to the student's advantage that he should receive his paediatric instruction on the neonate during the time he is studying obstetrics. Where practicable, it is equally desirable that study of the newly born should constitute an introduction to the wider subject of paediatrics and child health and should precede, not follow, other instruction given in general paediatrics.

ANTIHISTAMINES IN NON-URTICARIAL DERMATOSES, AND A RETRIAL OF ANTIHISTAMINE IONTOPHORESIS

BY

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AND

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In itching dermatoses other than urticaria and angioneurotic oedema oral treatment by the antihistamine drugs is attended by scant success. What slight improvement results is no greater than might be expected from the sedative action of the drugs alone. From America there are conflicting reports of the results of the use of antihistamine ointments. Thus Madden (1950) reports that of 141 patients with various dermatoses who were treated by phenindamine ("thephorin") ointment, 90% experienced relief or complete cure of the itching, and 5% were made worse. Wooldridge and Joseph (1949) report favourably on the use of phenindamine phosphate in ointment form, and consider that this substance penetrates the skin better than does diphenhydramine hydrochloride ("benadryl") or tripelennamine hydrochloride ("pyribenzamine"). On the other hand, Baldridge (1951), using phenindamine ointment on 41 clinically controlled cases, found that a 5% ointment produced no more relief from itching than did either of the ointment bases used—"carbowax 1500" and a vanishing-cream base. Furthermore, Ellis and Bundick (1949), using 5% phenindamine ointment in a carbowax base or as a 5% lotion in treatment of 50 chronic cases of dermatosis, found clinical sensitivity develop in 28% after an average of 50 days' treatment. They attribute the high incidence of sensitivity to the long period of use of the ointments, but they rightly point out that treatment is long-continued in many chronic dermatoses.

Similarly, McGavack *et al.* (1948) treated with benadryl ointment 74 patients suffering from itching dermatoses. They found complete relief from itching in 59.5% and improvement in 24.3%. They state that six patients felt discomfort from the applications—four of a temporary nature, the other two developing a weeping eczematous sensitization. But Perry (1947) found that the local use of benadryl ointment was of no value in neurodermatitis and of debatable value in other diseases.

After the disasters following application of sulphonamides and penicillin to the skin, English dermatologists seem to have made a more cautious approach to topical application of antihistaminics. Thus Warin (1950) stated that he had had little experience of external application of the antihistamine drugs. He presumed their main effect would be through their local analgesic properties. He felt that dermatitis had been provoked or aggravated by their use. Brain (1950) felt that "creams containing the antihistamine substances are occasionally of value, and they may be tried when other attempts at relieving the irritation of localized lesions fail."

Theoretically it is not to be expected that antihistamine therapy will be as effective in the treatment of eczema and dermatitis, where the site of reactivity is the epidermis, as they are in the case of the urticarial group of eruptions, in which the site of reactivity is the dermal papillae. It is probable that the antihistamine drugs act by blocking tissue receptors of histamine. In urticaria the antihistamine drug given orally is able to block the papillary receptors before the histamine from the epidermis is taken up in the papillae. In the case, however, of dermatitis or eczema the histamine is liberated in the target organ, in this case the epidermis, so that the antihistamine drugs will have less opportunity of blocking tissue receptors of histamine. There is, however, evidence that these drugs have a deterrent action on the actual liberation of histamine by allergic and other types of reaction which normally cause its appearance (Dale, 1950). If this is true the antihistamine drugs might be of use, provided they can be introduced into the epidermis.

Peck *et al.* (1950) found that it was possible to make 3 g. of ointment disappear if it was massaged into an area of skin 10 cm. in diameter. They used 2% pyribenzamine hydrochloride in a petrolatum base and rubbed it into normal skin. By estimating the urinary excretion of pyribenzamine they found that no appreciable quantity of the substance had penetrated the normal epidermal barrier. The same workers repeated the rubbing of ointment over areas of skin which were affected by dermatitis. They found that in a patient suffering from contact dermatitis 2% of the pyribenzamine contained in the ointment was excreted in the urine. They concluded that topical applications of antihistaminics readily found their way through the damaged epidermis, and seemed to be the logical mode of administration of antihistaminics for the treatment of dermatitis. Aaron, Peck, and Abramson (1948), however, state: "Recently ointments containing histamine antagonists for use in dermatitis have been under study. The results were not satisfactory in most instances." Possibly part of the explanation of the failure of the antihistamine ointments is to be found in the violence which is required to cause the antihistamine substances to penetrate the epidermis. Thus it is not surprising that any topical therapy which requires rubbing into areas of dermatitis for five minutes will do more harm than good. In view of this it was felt that a retrial of antihistamine iontophoresis as performed by Aaron, Peck, and Abramson would be worth while, since this method would ensure penetration of the epidermis by the antihistamine compound without the trauma of the rubbing required with the use of antihistamine ointments.

Choice of Antihistaminic Substance for Iontophoresis

Shelley, McConahy, and Hesbacher (1950) introduced by iontophoresis nine antihistaminic compounds into the skin of the back of five normal male subjects. Five minutes later histamine was introduced iontophoretically into the test areas. By the grading of the whealing so produced they assessed the efficiency of the antihistamines under test. They found pyribenzamine, "diatrin," and mepyramine hydrogen maleate ("neoantergan," "anthisan") the most effective in histamine wheal suppression. They quote a similar study by Rasmussen (1949), who found anthisan, promethazine hydrochloride ("phenergan"), and pyribenzamine the most active. Since no compound was found more active than pyribenzamine it was selected for clinical retrial, after the method of Aaron, Peck, and Abramson.

Selection of Cases

It was felt that only localized areas of skin were suitable for treatment, so that cases of extensive dermatitis were excluded from the test. So far as was possible, cases of

considerable chronicity were chosen, since a beneficial effect on a dermatosis of recent origin would be less significant. Very acute or weeping eczemas were excluded, because Aaron, Peck, and Abramson (1948) report that their patients experienced a burning sensation about one hour after treatment.

Method

A single thickness of lint soaked in a 5% solution of pyribenzamine was placed in contact with a pliable zinc anode cut to the shape of the lesion, and both were then covered by a pad of turkish towelling soaked in 2% saline. The anode so constituted was kept in place by rubber bandages and sandbags. The smallest electrode used was 15 cm.^{2} and the largest 100 cm.^{2} The indifferent electrode was generally attached to the sacrum. The current density varied between 0.08 and 0.25 mA per cm.^{2} Whereas Aaron, Peck, and Abramson employed daily treatments of five minutes for seven to ten days, treatment three times weekly was preferred. Also, the duration of treatment was not limited to five minutes, but in most cases was gradually increased up to a maximum of 20 minutes.

Results

The results are listed in the Table. Improvement is classified as subjective and objective: 0=no improvement; +=slight improvement; ++=moderate improvement; +++=great improvement; ++++=complete absence of itching or complete disappearance of visible lesions. If the patient was inconsistent in his statements or the estimate of improvement differed between us the least estimated improvement was tabulated.

Of the 20 cases listed 19 were very itchy lesions and one (Case 10) never had irritation. Of these 19, nine were either completely relieved of irritation or nearly so, one was moderately relieved, and in one (Case 17) it was not possible to evaluate the itching sensation. The remaining eight cases were not materially relieved of itching. In Case 15 the itching was worse after treatment and the skin looked worse. This patient, a man with undescended testicles and an unusual home environment, had eczema with marked endocrine defect. Case 1, lichen sclerosus et atrophicus, in which the diagnosis was confirmed by biopsy, was unusual in the intensity of the vulval itching, which had for some months almost driven her to distraction. It had resisted all the usual treatments except *x*-ray therapy, which was regarded as unsuitable for such an atrophic condition. Of the 20 cases, only five were much improved objectively, five others being moderately improved, and one made worse.

Toxic Effects.—Only Case 7 complained of an uncomfortable burning sensation about three-quarters of an hour after each treatment. There were no other toxic effects in this series.

Follow-up.—These cases were followed up some 12 to 18 months after the course of pyribenzamine iontophoresis. Of the 10 cases which had been improved by the pyribenzamine treatment four have remained entirely free from symptoms without further treatment (Cases 4, 5, 12, and 19); five (Cases 1, 3, 6, 8, and 14) relapsed slightly but remained substantially better than before the treatment; one (Case 16) relapsed absolutely. Case 17, in which the itching sensation had not been assessable, has remained entirely free from skin eruption without any further treatment. Of the eight cases not improved by the treatment one (Case 18) showed much improvement after a second course of the pyribenzamine iontophoresis; one was fatal (Case 11—carcinoma of the lung); three have remained unchanged in spite of further and different treatments (Cases 7, 13, and 20); and three (Cases 2, 9, and 15) have been cleared or greatly improved by other means, including prolonged in-patient treatment in Cases 9 and 15. The case of dermatitis repens (Case 10) has somewhat improved following many other and varied treatments.

Comment

Of the 20 cases treated by pyribenzamine iontophoresis Case 1 (lichen sclerosus et atrophicus) is probably an organic skin disease.

Table of Results

Case No.	Sex and Age	Type and Duration of Skin Lesion	Treatment before Iontophoresis and its Result	No of Treatments	Improvement	
					Subjective	Objective
1	F 59	Lichen sclerosus et atrophicus chest and vulva, with severe vulval itch for 4 months	Failure of rest, sedatives, tar, and carbolic lotions to give any relief	15	+++	0
2	F 47	15 years' 4½ by 3½ in. (10·3 by 8·8 cm.) patch eczema outer right ankle	Failure of internal saphenous ligature and injection of moderate calf varicosities. Failure of subsequent *x*-ray therapy and usual applications	13	0	0
3	F 64	15 years' 3 by 4 in. (7·5 by 10 cm.) patch of thickened neurodermatitis below left knee	None known	12	+++	++
4	F 34	3 years' lichenified eczema right supraclavicular area and right side neck	*X*-ray therapy with sedatives and antipruritics cleared it temporarily; recurrence 5 months later	7	+++	+++
5	F 53	Prurigo of inner thighs and groins, with high blood pressure	Temporary clearance with *x*-ray therapy and applications. Recurrence	19	+++	++++
6	F 32	Right supraclavicular neurodermatitis. 9 months	None	14	++	++
7	F 13	4 years' patch infected recurrent eczema inner wrist	Gentian violet ointment	11	0	0
8	F 37	3 years' neurodermatitis of nape	None	14	++++	+++
9	M 55	Chronic patch eczema back of left leg "present most of his life"	None but biopsy, first performed to confirm diagnosis	9	+	0
10	M 44	Dermatitis repens left lower leg. Some months	Oral sulphonamides and parenteral penicillin besides usual skin antiseptic applications	13	—	++
11	M 58	5 years' thickened scrotal eczema	Many and varied over 2 years without success	15	0	0
12	M 43	15 years' continuous pubic neurodermatitis	Partial temporary relief by *x*-ray therapy followed by relapse	13	+++	+++
13	M 38	20 years' patch of eczema behind left knee	None	15	0	+
14	M 41	5 years' symmetrical thickened eczema about upper end of intergluteal cleft	Recurrence 6 months after *x*-ray therapy	22	+++	++
15	M 38	10 by 5 in. (25 by 10 cm.) left upper calf, lichenified thickened patch eczema some months	Only temporary relief from itching by *x* rays	4	Worse	Worse
16	M 29	8 months' seborrhoeic folliculitis of nape	None	12	++++	0
17	M 8	Patchy excoriated eczema inner and outer left ankle 3 months	None	24	?	+++
18	M 30	3½ years' pruritus ani; anal skin white, thick, and sodden	*X*-ray therapy had produced improvement with lotion and advice	23	++	0
19	M 72	6 months' papular eczema of wrist following scald		20	++++	++
20	M 41	9 weeks' pruritus ani with anal skin tags and some stenosis following pile operation	None, except stopping of liquid paraffin without improvement	13	+	+

In assessing the prognosis in any case of eczema or other functional itching skin disorder a large number of factors require consideration. Factors prejudicial to recovery might be grouped thus:

1. *Psychological.*—Domestic disharmony, frustration, or unhappiness at work. Overcrowded housing conditions and friction with "in-laws." Overwork, worry, insomnia, compensation disputes.

2. *General.*—Hypertension, anaemia, dyspnoea from chronic bronchitis, emphysema, or other cause. Alcoholism. Poor renal function as in prostatic obstruction and other states of sub-health. Faulty nutrition. Bad heredity.

3. *Endocrine State.*—Unfavourable influence of puberty and menopause.

4. *Chronicity of the Lesions.*—A dermatosis of many years' duration is likely to be more intractable than one of months or weeks.

5. *Local Factors* (e.g., xerodermia or hyperidrosis).—Haemorrhoids or fistula in pruritus ani, ankle oedema due to varicose veins, deep thrombosis or other cause in leg eczema. Perniosis. Impossibility of avoiding local allergic contacts.

6. Unwillingness or inability to carry out exact treatment.

In attempting to estimate the worth or otherwise of any therapy in these conditions much reliance must be placed on clinical judgment, since cases exactly comparable in every respect with regard to such factors as those mentioned above are not available.

Summary and Conclusion

Although antihistamine ointments can be absorbed when massaged into areas of dermatitis the friction and trauma of the rubbing may make the dermatitis worse. Their use is therefore likely to be limited, and any beneficial effect is probably due to their gentle application as a surface analgesic. Since they are also potent sensitizers they should be used, for short periods only, on localized areas of skin. They are seldom used in the Sunderland Skin Clinic, and several cases of dermatitis due to them have been encountered.

The introduction of antihistamines into the skin by iontophoresis can be brought about without the trauma and friction necessary to introduce the ointment. The method is free from toxic reactions, and for localized areas of chronic dermatitis or pruritus may have a useful though limited role.

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Two hundred years ago on May 19 Antonio Scarpa, the Italian anatomist and surgeon, was born. He studied under Morgagni and others at Padua, and became an expert anatomist. Soon after obtaining his degree in medicine he published an important work on the ear, in which he recorded his discovery of the membranous labyrinth. During a visit to London he worked with Pott and the Hunters. As professor of anatomy and surgery at Pavia he built his reputation as the greatest Italian anatomist and surgeon of his century. His *Tabulae Neurologicae*, 1794, with beautiful drawings by Scarpa himself, showed him to be one of the best of those medical men who have illustrated their own works. He was the first to tie the femoral artery for popliteal aneurysm. Scarpa was blind for the last few years of his life and died on October 31, 1832.

A CASE OF CONFLUENT CHICKEN-POX WITH HAEMORRHAGIC SYMPTOMS

BY

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AND

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The following is a record of a case of chicken-pox of exceptional severity which exhibited a confluent haemorrhagic eruption and a severe leucopenia. Recovery followed treatment with "aureomycin."

Case Report

The patient was a man aged 40, hitherto in good health. On July 21 a small boy arrived to stay in the patient's house, but was found to be suffering from chicken-pox and returned to his own home on July 25. So far as is known there were no unusual features about this attack. Two children of our patient, aged 10 and 4 years, developed chicken-pox about August 3. We are informed that each experienced a mild attack and made a rapid recovery. The patient felt unwell on August 4 and a rash began to appear next day. When first seen by his doctor, on August 7, "he had a very extensive varicella rash and what looked like a follicular tonsillitis": he was febrile, and was sent home to bed and put on sulphonamides. On August 9, as he was vomiting after a restless night, the drug was omitted. The temperature then was 101° F. (38.3° C.). At night he was kept awake by persistent hiccup. On August 10 his temperature had risen still further and the rash began to show a purpuric element: 400,000 units of penicillin was given. Hiccup returned at night. On August 11 aureomycin therapy was started (0.5 g. six-hourly) and admission to hospital was sought because of the severity of the illness and the need for investigation of the haemorrhagic symptoms. Attempts to obtain suitable hospital accommodation nearer his home having failed, we agreed, on his doctor's advice, to admit him to our unit, although this entailed a journey of some 50 miles (80 km.) up from the country.

On arrival the patient was found to have an extremely profuse eruption (Figs. 1 and 2), confluent over the face and on parts of the trunk, quite generalized, and exhibiting no selectivity except that the profusion was greatest on the trunk and face. The rash generally showed purpuric staining, but there was, at this stage, no free bleeding into any of the many large superficial vesicles. The rash was mixed as regards age and size of its elements: it was pustular on the face, of which there was pronounced swelling. A profuse enanthem was present in the mouth and fauces, and there were lesions also on the conjunctivae. The patient felt weak and tired, the pulse was running, some rhonchi were

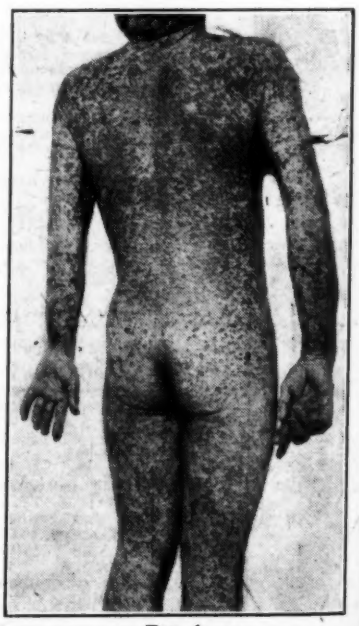

FIG. 1.

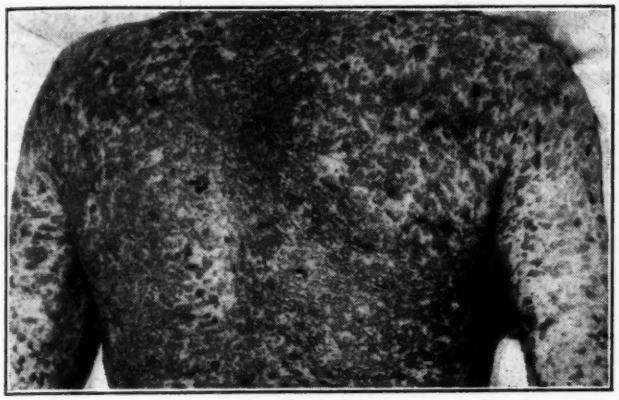

FIG. 2.

audible at the bases and his temperature was 101° F. (38.3° C.). The dosage of aureomycin was increased to 0.5 g. four-hourly. He remained toxic, breathless, and exhausted on any exertion; the temperature rose to 103° F. (39.4° C.) and the pulse to 125. Hiccup was especially persistent at night, when sleep could be obtained only by the use of morphine. The rash progressed irregularly. No free bleeding occurred into the skin or from mucous surfaces, but there was widespread purpuric staining of the elements of the rash and more obvious haemorrhage at the base of and into some vesicles. Gradual improvement followed: the fever had subsided by August 17, when the aureomycin treatment was stopped. On August 21, when many of the crusts were separating and all anxiety regarding his recovery had disappeared, the patient returned, at his own wish, to his home environment. Styes and boils caused some trouble in convalescence.

Investigations.—On August 13 the vesicle fluid was negative to the complement fixation test for the vaccinia-variola group; egg culture was negative for vaccinia and variola. A throat swab showed no Vincent's organisms present in film. *Strep. viridans* and yeast were found on culture. The blood picture on August 13 was: Hb, 104%; red cells appear normal; platelets moderately diminished; white cells, 1,500 per c.mm. (polymorphs 57%, lymphocytes 39%, monocytes 2%, plasma cells 2%). On August 14: white cells 4,200 per c.mm. (polymorphs 54%, lymphocytes 43%, monocytes 3%). On August 16: white cells, 5,500 per c.mm. (polymorphs 61%, lymphocytes 33%, monocytes 5%, plasma cells 1%).

Discussion

An attack of varicella of the severity shown in this case must be extremely rare. During the past 23 years we have personally examined some 400 chicken-pox patients in whom, often because of the profusion of the rash or of the amount of constitutional disturbance, a diagnosis of smallpox has been considered. Yet among these severe cases we can recall only two, one of which proved fatal, which were comparable to the one here described. That the type of attack in this instance was not determined by an especially virulent strain of virus is suggested by the mildness of the disease in the three children who were known to be infected in the same series.

We have hesitated to apply the term "haemorrhagic" to this case as, following the teaching of Ricketts (1908) in respect of smallpox, we would reserve its use, in any acute infection, to describe the type of case in which initial toxaemia is extreme, and usually rapidly fatal, and in which considerable skin and mucous membrane bleeding may be prominent. In spite of the fact that the present patient's condition gave rise to some anxiety, we do not consider that his case falls into this category.

It has been suggested by M. Mitman (1951, personal communication) that in some instances haemorrhagic symptoms in chicken-pox may be determined by the occurrence of this disease in a subject already afflicted with some disorder of the blood. In the above case a marked leucopenia at the first blood examination, combined with the severe faucial angina, pointed to an agranulocytosis, possibly determined by idiosyncrasy to sulphonamides. However, in spite of the low total white count, the polymorphonuclear cells never fell below 54%, and in the course of a few days the blood picture resumed a more normal appearance, in keeping with the general clinical improvement of the patient. Consequently we regarded the leucopenia as a symptom and not as a cause of the unexplained lack of resistance to the virus which determined the severity of the attack.

The enanthem formed a serious feature. The mouth and the fauces, for as far as they could be inspected, were profusely involved. The cause of the hiccup may well have come from lesions more remotely situated. But for antibiotic therapy the risk of pulmonary complications must have been considerable.

We wish to thank Dr. J. A. J. Hammond, who referred the case to our unit, for details of the patient's illness before his admission to hospital.

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A CASE OF ACCIDENTAL GENERALIZED VACCINIA

BY

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The following case of accidental generalized vaccinia demonstrates the source of infection and presents unusually extensive pathological lesions.

Case Report

A male child aged 6 months and weighing 16 lb. (7.3 kg.) was admitted on June 23, 1948, with exudative infantile eczema affecting the face, buttocks, and outer aspects of the thighs. Next day it was discovered that a female child who had been admitted 13 hours previously to a cot adjacent to the patient's had an active vaccination sore. This child was immediately isolated and made an uneventful recovery.

The patient responded satisfactorily to treatment, but 12 days later a number of papulo-vesicles appeared on the buttocks and thighs. The lesions, which rapidly became pustular, were discrete, umbilicated, roughly circular, of similar size, and at the same stage of evolution. With this exanthem the child became febrile and penicillin therapy was instituted. Two days later further crops of vesicles appeared on the face, trunk, and limbs, but no pulmonary lesion was detected clinically or on *x*-ray examination. By July 11 most of the pustular lesions had become small ulcers, several of which had coalesced, producing large ulcerated areas (Fig. 1) associated with inguinal and axillary adenitis. Sulphadiazine therapy was started. On July 14, for the first time, there was reluctance to feed and facial grimacing became a marked feature: signs of bronchopneumonia were now present. Death occurred on July 15.

Post-mortem Findings

The necropsy (four hours after death) showed lesions only in the skin (see above), lungs, and stomach.

FIG. 1.—Generalized vaccinia: eruption over sacrum, buttocks, and lateral side of thigh. Taken on July 13, 1948.

Lungs.—Small firm dark-purple nodules, a few with central vesiculation, were scattered on the surface of the right middle and both lower lobes. Similar nodules were present in the substance of these lobes, each consisting of a dark-purple consolidated area (approximately 0.5 cm. in diameter) with a grey or yellowish centre (0.1 cm. to 0.2 cm. in diameter). Altogether, 12 to 15 such nodules occurred in the three lobes.

Stomach.—The mucous membrane showed several dark-red areas of varying size, in which numbers of small ulcers were present.

Histology

Skin.—The appearances of the lesions were typical at all stages, and only certain features will be described, for comparison with those in the lungs. In the pustular stage, cellular infiltration was a prominent feature, both in the centre of the lesion and at the edges, where small vesicles were still present in the epidermis (Fig. 2): the cells were mononuclears, lymphocytes, macrophages, and endothelioid; polymorphs and eosinophils were scanty. Congestion and small haemorrhages were present. In the more advanced pustules small areas of necrosis were seen in the superficial part of the cutis vera. In the sections of all lesions, elementary bodies were very numerous. They were most clearly seen in the cytoplasm of the oedematous cells at the edges of vesicles (Fig. 3), where they occurred singly, in pairs, or in small groups mostly coccoid or rod-shaped, though varying somewhat in shape and size. Under serous crusts of vesicles, the bodies were very numerous and massed together; bacteria were also present. In the floors of pustules and ulcers, necrotic cellular debris made identification of the bodies uncertain, though it appeared that foci of large numbers were present. In contrast to the large number of elementary bodies, inclusion bodies of the Guarnieri type were scanty. None were seen in sections of vesicles. A few were present in epithelial cells at the edge of a pustule.

FIG. 2.—Skin: edge of pustule. Multiple vesicle formation with subepidermal oedema and extensive cellular infiltration. (H. and E. ×120.)

FIG. 3.—Skin: early lesion. Elementary bodies in oedematous epithelium at edge of lesion. (Castaneda. ×600.)

Lungs.—The nodular areas showed a histological structure similar to each other and comparable to the skin lesions. The nodules with clear grey centres showed discrete dense areas with an oedematous convexity towards the pleural surface similar to the late vesicular stage in the skin. Where the centre was dull yellow, necrosis was present, corresponding to the pustule. In both degrees of the lesion the surrounding lung tissue showed only slight oedema: the bronchioles both outside and just within the dense area showed no evidence of pyogenic or any other inflammatory changes. In the alveoli of the affected areas there was oedema with intense congestion of the capillaries in the walls; both the alveoli and the interstitial tissue showed extensive cellular infiltration similar to that found in the pustular skin lesions. In all the lesions numerous elementary bodies were present, in masses towards the pleural surface (Fig. 4) and scattered throughout the lesion, where the occurrence of a few Gram-positive diplococci emphasized the size of the elementary bodies. Guarnieri inclusion bodies were not seen.

Stomach.—The most obvious change was a punched-out ulcer of the mucous membrane, sometimes involving the muscularis mucosa and part of the submucosa. There was extensive oedema corresponding to vesicle formation in the submucosa with early necrosis of the collagen fibres. In one submucous "vesicle" small clumps of a few elementary bodies were attached to a necrotic collagen fibre.

Discussion

This child, suffering from infantile eczema, developed generalized vaccinia following contact with another child who had been vaccinated 10 days previously. Contact consisted merely in the occupation by the children of neighbouring cots for a period of 13 hours, mostly during the night: the first elements of the generalized vaccinia became evident 12 days later. Harries and Mitman's (1947) report that generalized vaccinia occurs especially among those who already have some skin condition was confirmed in a review of previous reports by Van Rooyen and Rhodes (1948, p. 391), and later by Fries *et al.* (1948), Anderson (1949), Whittle *et al.* (1950), and Sommerville *et al.* (1951).

While similar cases are often reported as Kaposi's varicelliform eruption, this term has been applied to clinical conditions due to

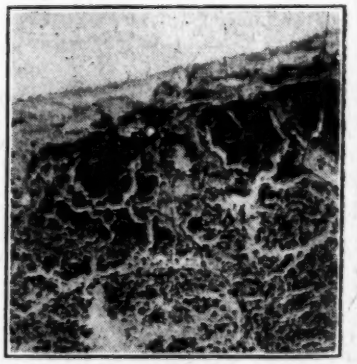

FIG. 4.—Lung lesion at pleural surface, showing large number of organisms massed together. (H. and E. ×120.)

different viruses—for example, vaccinia (Anderson, 1949; Whittle *et al.*, 1950), herpes simplex (Jaquette *et al.*, 1946; Brain *et al.*, 1950), possibly influenza (Leverton and Whitlock, 1949)—or in which the virus is not definitely known (cf. Van Rooyen and Rhodes, 1948, p. 299). It would seem reasonable to apply the more specific term of "accidental generalized vaccinia" or "Kaposi's varicelliform eruption (vaccinial type)" to cases in which the infection (*a*) is acquired by contact with a vaccinated person, (*b*) is definitely due to the vaccinia virus, and (*c*) causes a pox eruption on an eczematous soil (Fries and Borne, 1949).

The unusual lung lesions showed the same basic pathological features as those in the skin. Elementary bodies were present in large numbers. Viral lesions in bronchial walls have been described in mice inoculated intranasally (Nelson, 1938) and in generalized vaccinia in rabbits (Douglas *et al.*, 1929; McIntosh and Scarff, 1929), but not in man. In spontaneous rabbit pox, on the other hand, lung lesions exactly similar to those described here have been depicted by Nicolau and Kopciowska (1931). While the lesions in the stomach were only at the early "vesicle" stage, and no cellular infiltration had developed, it is assumed that they also were primarily due to the presence of the virus elementary bodies.

Large numbers of cytoplasmic and intercellular elementary bodies—Buist or Paschen type (Gordon, 1937)—occur in the skin lesions of vaccinia, but, so far as can be ascertained, lung and stomach lesions containing these bodies have not been previously reported in man. The large eosinophilic inclusion bodies of Guarnieri are specific; in this case they were seen only in the skin lesions, and not in those occurring in the lungs or stomach.

Summary

A fatal case of accidental generalized vaccinia is described; the clinical history lays fresh emphasis on the ease with which an eczematous infant may develop generalized vaccinia. In addition to the skin eruption, lesions of a similar nature were present in the lungs and stomach.

We are indebted to Drs. A. D. McLachlan, and A. Girdwood Fergusson, Department of Dermatology, Stobhill Hospital, for their advice in the presentation of this paper.

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People sometimes do too much to help the blind, and increase their feeling of helplessness. Unwittingly they leave too little of the initiative with the blind person, says the *Annual Report* of the Guide Dogs for the Blind Association. But the guide dogs treat their masters as normal in everything except that they cannot see. They guide their masters, but do not think for them. Dogs enable many people to do jobs that would otherwise be extremely difficult for them—those involving travelling, for instance. For hundreds of years people have used dogs to guide them, but not until 1915 was any organized training begun. This was in Germany, and the British association was founded in 1933. In addition to the centre at Leamington, a new one has been set up at Exeter.

Medical Memoranda

A Case of Mumps with Involvement of the Lacrimal Glands

Involvement of the lacrimal glands in mumps must be very rare, although reference to it can be found in some standard works: "The lacrimal gland is rarely attacked" (Price, 1950) and "Vaugiraud and Defaux have reported a case of bilateral dacroadenitis manifested by swelling and tenderness of the lacrimal glands, which developed three days after the parotitis and subsided at the same time as the inflammation of the parotid" (Rolleston and Ronaldson, 1940). Nevertheless it was with great surprise that we encountered the following case in which an undoubted bilateral dacroadenitis coexisted with, and clearly formed an integral part of, an otherwise typical mumps syndrome.

CASE REPORT

On March 18, 1951, a woman aged 29 complained of general aches, malaise, and pains in the left side of the neck. Next day the aches had disappeared and she was able to do her housework in the morning. The pain in her neck persisted, however; in addition her left upper eyelid began to swell and she became conscious of two swellings, which she could feel with the tip of her tongue, in the position of the sublingual glands. By the same evening she had developed a frontal headache, which she described as very severe and "more than just a headache." At this stage her neck felt "stiff" and her left upper eyelid began to swell. On March 20 the frontal headache remained severe, both upper eyelids were very swollen (but without erythema), and the neck was painful on both sides and diffusely "stiff." The patient remained conscious of the swellings under her tongue. At this time the oedematous upper eyelids were the outstanding sign and the possibility of nephritis was considered.

On March 21 the eyes were almost closed by oedema and a notable erythema involved the lids of both eyes, ceasing abruptly at the eyebrows and spreading downwards on to the cheeks, where it faded away without line of demarcation. The erythema did not straddle the nose or involve the forehead or neck, and it was not indurated or tender. The headache had by now disappeared. The pain in the neck remained, and there was also pain in both ears and underneath the right upper eyelid. On this day the patient was admitted to hospital as a presumptive case of facial erysipelas. Besides the facial appearances, tender smooth enlargements of moderate degree were found discretely affecting the parotid and submaxillary glands of both sides; there was also a moderate degree of trismus, and the tongue could not be protruded more than just to show the tip—owing, it appeared, partly to the mechanical effect of the sublingual swellings and partly to their tenderness. The dorsum of the tongue was dry and brownish and there was pronounced halitosis. The temperature on admission was 100.4° F. (38° C.) and the urine was not found to contain albumin or sugar. On March 22 the patient began to feel better, the temperature fell in the evening (subsequently remaining normal) and the oedema of the eyelids began to decrease.

On March 23 a blood count gave haemoglobin 90%; colour index, 1; red cells, 4,500,000; white cells, 9,500 (polymorphs 75%, lymphocytes 20%, large monocytes 4%, eosinophils 1%). On this day the upper eyelids could easily be lifted and a swelling could be discerned under the outer part of each; pressure on this swelling through the lid definitely caused pain—at least on the right side—and the examining finger left a pit in the subsiding oedema. Underneath each upper lid was a small amount of sticky yellowish exudate. The patient was seen by an ophthalmologist, Mrs. Dorothy Campbell, on March 23, who noted: "Acute

adenitis of lacrimal glands with local conjunctival injection. Sclerae white. No signs of iritis." Rapid recovery ensued, and by March 28 all symptoms and signs had disappeared.

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An Unusual Case of Acute Pancreatitis

The following case of a woman aged 39 who died of acute pancreatitis on the 37th day of her disease presented two unusual features: (1) the appearance, in the early stage of the disease, of a syndrome which was indistinguishable from diabetes mellitus; and (2) death from intra-abdominal haemorrhage.

CASE REPORT

The patient was admitted to hospital on January 9, 1950, with the following history. For two years she had been subject to repeated attacks of severe epigastric pain with some flatulence, the symptoms being unrelated to food and not relieved by taking more food or alkaline powders. On January 5 she had an extremely severe attack of this indigestion. There were no further definite symptoms, although she felt unwell, until three days later, when, after a normal bowel motion, she developed severe upper abdominal pain and vomited. The pain and vomiting continued until her admission. She admitted to some backache when questioned, but could not localize it to either the lumbar or the interscapular region. When the urine was found to be loaded with sugar and acetone she was questioned about a history of loss of weight, of polyuria, or of polydipsia. She denied ever having had these symptoms.

On examination she was pale, anxious, and moderately shocked: blood pressure 130/105, temperature 98° F. (36.7° C.), pulse 120, respirations 25. Her tongue was dry and furred. The normal respiratory movements of the abdomen were diminished; there was tenderness and guarding over the upper abdomen and slight tenderness over the lower abdomen; she felt a sharp pain on the release of pressure over the whole abdomen; there was no diminution of liver dullness; the abdomen was silent on auscultation. Rectal examination disclosed acute tenderness in the pouch of Douglas. Examination of the respiratory and cardiovascular systems revealed no abnormality. Urine: brick-red with Benedict's solution; acetone +++ (Rothera's test); chlorides, 3 g. per litre (Marriott's test); diastatic index (result not received until after laparotomy), 250 units. Blood chemistry: sugar, 258 mg. per 100 ml.; urea, 93 mg. per 100 ml.; chlorides, 468 mg. per 100 ml.; protein, 5 g. per 100 ml. Radiography showed no evidence of gas under the diaphragm.

Because the clinical features were so much more like a perforated peptic ulcer than an acute pancreatitis and the diastatic index was not available, it was decided to do a laparotomy. An intravenous infusion of normal saline was started, and 10 units of soluble insulin was given together with 30 g. of glucose intravenously. On opening the peritoneal cavity sero-sanguineous fluid escaped. Numerous areas of fat necrosis were seen on the peritoneum and the great omentum. The gall-bladder contained at least one large calculus. A diagnosis of acute pancreatitis having been made, a drain was placed in the lesser sac and the incision closed.

The patient made satisfactory progress for the first 10 days. On the morning after operation the blood-sugar level had fallen to 153 mg. per 100 ml. A further dose of 20 units of soluble insulin covered by 50 g. of glucose intravenously was given. The urine was tested with Benedict's solution every four hours, and, apart from one orange result on the third post-operative day (this was associated with a blood-sugar level of 267 mg. per 100 ml.), the test persistently gave a green or blue result. A glucose-tolerance test was done on the 13th day after operation. The result was as follows:

Time	*Blood-sugar Level*	*Urine*
Fasting	90 mg. per 100 ml.	No sugar
½ hour	175 " " " "	" "
1 "	205 " " " "	Sugar +++
1½ hours	174 " " " "	" +++
2 "	202 " " " "	" +++

The whole-blood chlorides remained within normal limits.

During the first week there was a purulent bronchitis, but up to the 10th post-operative day the patient's general condition gradually improved. On this day a thick yellow discharge from the drain began, and she started to go downhill. She lost her appetite and began to waste rapidly. The nature of the discharge soon altered, portions of necrotic pancreas being seen. The discharge began to digest the abdominal wall. Continuous suction was applied to the drain, and this proved very effective in protecting the abdominal wall. A severe anaemia was associated with the rapid wasting. On the 14th post-operative day the haemoglobin was 10.56 g. per 100 ml. (72%). There was no response to iron, liver, or transfusion, and her condition deteriorated rapidly. Thirty-seven days after the onset of the acute illness dark blood began to pour from the drainage tube. A blood transfusion was begun, and she received 3 litres of stored blood. She died six hours after the haemorrhage began.

Post-mortem examination showed thick blood-stained fluid and clots in the peritoneal cavity, a plastic peritonitis involving particularly the small intestine, fat necrosis of the greater omentum, and disintegration of the left half of the pancreas. Histological examination of the surviving portion of the pancreas showed the normal architecture to be quite well preserved; groups of acini showed disorganization and autolysis, but there was no haemorrhagic infiltration. The pancreatitis in this situation appeared to be resolving. The gall-bladder contained a large stone and several smaller calculi; its wall was moderately thickened.

COMMENT

The interest in this case arises not from the presenting symptoms and signs but from the complications.

Although it is not uncommon for there to be a slight glycosuria, so far as can be ascertained it is uncommon for a severe glycosuria and hyperglycaemia to be found. There can be no doubt that before the onset of the acute pancreatitis this patient had no symptoms to suggest that she suffered from severe diabetes mellitus and it seems unlikely that she had even mild diabetes. Yet 36 hours after the onset of the acute illness her urine was loaded with sugar and acetone and her blood-sugar level was considerably raised. This diabetic state slowly subsided and her blood-sugar level and urine were normal by the 12th post-operative day. Next day the glucose-tolerance test was abnormal. The pathogenesis of this phenomenon was probably a complete temporary inhibition of the islet tissue due to the action of the escaping enzymes. The islet tissues then gradually recovered, though incompletely. It is well known that mild diabetes mellitus may follow recovery from acute pancreatitis. It is interesting to note that although this was a severe case of the disease the histological examination of the pancreas showed many areas of apparently normal pancreatic tissue.

I do not know of any other case of acute pancreatitis in which haemorrhage from a splenic vessel was the cause of death, but it cannot be considered a surprising complication. It must be presumed, in spite of the histological findings, that the disease was still active, that the enzyme was still being liberated, and that this digested the wall of the vessel.

I wish to thank Mr. Michael Harmer and Dr. A. Piney for their advice and criticism and Dr. F. Woodhouse-Price and Dr. M. S. Ross for the post-mortem details.

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Reviews

ASTLEY COOPER

The Life and Work of Astley Cooper. By R. C. Brock, M.S., F.R.C.S. (Pp. 176; illustrated. £1.) Edinburgh and London: E. and S. Livingstone. 1952.

The name of Astley Cooper, one of the really great surgeons of the early nineteenth century, is held in honour by us all, and his magnificent portrait by Lawrence is one of the treasures of the Royal College of Surgeons. The pupil of Henry Cline and of John Hunter, like them he based his work on the study of anatomy, and like them he was a man of boundless energy and of tireless industry. The founder of Guy's Hospital Medical School, it is appropriate that his life should have been written by a Guy's surgeon, but that the writer should be at the same time a leader in arterial surgery is singularly fortunate. For it was in the surgery of the arteries that Astley Cooper achieved his greatest fame, and his courage and skill in that difficult field long ago must win our admiration.

Mr. Brock has produced a fascinating study of his great predecessor, a study full of human interest and also of deep surgical significance. He has painted a vivid portrait of the life of a busy surgeon in days when anaesthesia and asepsis were both unknown and in a society which has long since vanished. It will be read with pleasure and interest as a story of courage, devotion, and high achievement, and there is much that we may still learn from the story so well told of a great surgeon and a great teacher.

HENRY SOUTTAR.

PAEDIATRIC UROLOGY

Clinical Pediatric Urology. By Meredith Campbell, M.S., M.D., F.A.C.S. With a section on Nephritis by Elvira Goettsch, M.D., and John D. Lyttle, M.D. (Pp. 1,136; 543 figures. £4 10s.) Philadelphia and London: W. B. Saunders Company. 1951.

This new version of Professor Meredith Campbell's well-known manual makes a book emphatically for the desk and not the fireside armchair. It is comprehensive, a veritable encyclopaedia of child urology, in which special field the author's experience and command of material must be unsurpassed. There is no conceivable anomaly of the child's urinary tract of which a full account is not be to be found in this book, usually illustrated by examples of actual cases from the author's practice. This ensures its abiding value as a work of reference. To the ordinary reader seeking guidance in more simple matters, however, it is less satisfying. Amid such profusion, perspective tends to be obscured by prolixity. Some pruning would be an improvement.

Hydrocele, hernia, and imperfect descent of the testis are not really "urology," and the operative procedures here described for them would seem to many to be needlessly complicated. The Torek type of orchidopexy must be rarely practised by children's surgeons in Britain. In the correction of hypospadias the author rightly gives precedence to the Denis Browne operation. It is to be hoped that the description of other more complicated procedures which is here included will not encourage their continued use. The author attributes meatal ulceration to congenital stenosis of the orifice; others experienced in these matters would certainly disagree with him in commending meatotomy as the essential treatment of this simple but distressing lesion.

In this comparatively new field of child urology, with so many problems still unsolved, differences of opinion are inevitable and indeed healthy. Readers with the requisite experience will not concur with all Professor Campbell's views as here expressed, but all would agree in congratulating him on the continued high standard of this excellent book, with its profusion of illustrations. The production is lavish, and the radiographic reproductions in particular are superb.

T. TWISTINGTON HIGGINS.

SKIN DISEASES

A Handbook of Diseases of the Skin. By Herbert O. Mackey, F.R.C.S.I., L.R.C.P.I., D.P.H., L.M., F.R.I.A.M. (Pp. 216; 36 illustrations. 7s. 6d.) Dublin: C. J. Fallon. 1952.

The author is to be congratulated on a good presentation of a difficult subject. It is particularly useful for the student because of its small compass and its ready interpretation in terms of general medicine. The book is in no way a potted version of dermatology divorced from the rest of practice, and for this reason it may be recommended to the busy practitioner. The presentation in the main is balanced, but a few faults should be corrected in further editions. Mention of some rare diseases should be omitted, while others such as lichen simplex and lupus erythematosus want more attention. Seborrhoeic dermatitis is not discussed in the text, though there are illustrations and a dozen references in the index. There are too many references in the index which lead nowhere.

The reviewer would like to see fewer differential diagnoses and theories of aetiology presented for the student rather than an uncritical mass of information in these sections. A little personal experience is much more educative. The account of infantile eczema—and the presentation of eczema as a whole—suffers a little from this defect, and sections otherwise excellent, such as those on varicose ulcers, impetigo, and fungous infections would be improved. Warty tuberculosis and pseudo-pelade are discussed twice, and pyodermia and lupus erythematosus should appear as common scarring diseases, while tinea versicolor should not appear as a papular eruption. Dr. Mackay has, however, succeeded in presenting a useful and brief account of present practice in dermatology, for which there must be a considerable demand.

J. T. INGRAM.

CARE OF THE DISABLED

Disabled Citizens. By Joan Simeon Clarke. Foreword by Lord Beveridge. (Pp. 237. 16s.) London: George Allen and Unwin. 1951.

The foreword by Lord Beveridge warns us that the picture painted by Mrs. Clarke is a grim one and that this book will be found to be neither pleasant nor cheerful. He is right. The author is stimulating as one expects a missionary and an enthusiast to be—and stimulation sometimes causes irritation. The volume is an attractive production, the arrangement is logical, and on the whole the writing is clear and well done, although I must confess that I do not know what is meant by "a scarcely imperceptible scoliosis." The index is a model of meticulous perfection.

The author does not appear to be satisfied that local authorities and their staffs are dealing adequately with the physically handicapped, and makes far-reaching proposals by means of which the care service in the homes would be much extended and some of the functions of the local authorities would be transferred. The welfare of the handicapped would be entrusted to a central body. Who would appoint the members is not clear. There would be local organizations covering the whole country and a case-work service would be conducted by trained social workers. Health visitors will not therefore be quite suitable for this work, because "they are not qualified social workers," according to Mrs. Clarke. The words quoted will cause surprise. One would have thought that for the care and aftercare of the disabled there could be no training even comparable to that of the health visitor, who in the course of her training spends three or four years nursing the disabled and a further year in intensive work in social service.

This book will certainly create discussion. That no doubt is one of the aims of the author. Those who wish to join in should provide themselves with the basis of the discussion by reading the book. I think, however, that the majority of those who practise preventive medicine and care and aftercare in the industrial town, where life is hard and

climate harsh, would consider that the difficult work of caring for the disabled is best entrusted to local authorities and their highly experienced staffs, who would certainly wish to have also the continued and valued help of voluntary organizations.

C. METCALFE BROWN.

CARBON DIOXIDE THERAPY

Carbon Dioxide Therapy. A Neurophysiological Treatment of Nervous Disorders. By L. J. Meduna, M.D. (Pp. 236. £1 16s.) Springfield, Illinois: Charles C. Thomas. Oxford: Blackwell Scientific Publications. 1950.

Carbon dioxide therapy is a recent addition to the abreactive methods of treatment in psychiatry. Inhalation of a mixture of 20 or 30% carbon dioxide with oxygen acts as an anaesthetic of rather the same type as ether. There is an induction phase when the patient tends to become excited, the phase of anaesthesia itself, and then the stage of recovery. During either the first or the last stage opportunity may be taken to use the clouded state of consciousness to induce the patient to react emotionally to the evocation of present worries or past memories, perhaps forgotten or repressed ones.

Dr. Meduna, the originator of this treatment as well as of the modern application of convulsive therapy, is inclined to overestimate its value. In Britain it has been used in a number of clinics and with varying techniques. Therapists are generally agreed that the patients who respond best to it are those who also respond to such similar methods as narco-analysis, ether abreaction, etc.—that is, patients whose symptoms combine anxiety and hysteria and are related to specific traumatic causes. In his book he describes at length the treatment of a psychopathic alcoholic addict, not the best type to choose for an example. Apart from this section, there are others on the physiological action of carbon dioxide, the technique of treatment, and a theory of its curative action based on the cybernetic views of McCulloch. There is no adequate discussion of indications for choice of this method, and the practical and clinical aspects are generally rather skimped. At the present stage of knowledge it is not possible to found carbon dioxide therapy, any more than the other so-called shock methods, on a secure theoretical basis, and we must still regard them as empirical.

ELIOT SLATER.

E.N.T. ATLAS

Atlas der Hals-Nasen-Ohren-Krankheiten. By Professor Carl von Eicken and A. S. van Treeck. Third enlarged edition. (Pp. 200; 470 figures. M. 78.) Stuttgart: Georg Thieme. 1951.

This atlas of throat, nose, and ear diseases was first published in 1940; a second edition was ready in 1943 but was damaged in the war, and the present edition is therefore the third. It is designed for students and practitioners to remind them of the anatomy, pathology, and diseases of the ear, nose, and throat, and in some cases operations are illustrated. The 470 illustrations are almost all coloured photographs and are extremely good. There is scarcely any disease that is not illustrated; all pictures are concisely described, and on the page facing them is a pithy account of the clinical picture, differential diagnosis, and treatment. The coloured photographs of the face of a number of patients showing various diseases are outstanding and would enable students and doctors to recognize the condition in their own patients. There is a description of the instruments used to photograph the tympanic membrane, the nasopharynx, and the larynx, and certainly the inventors are to be congratulated on the results achieved. At the head of each section is a picture showing how to examine the part under review and a comprehensive account of the anatomy explained by clear diagrams and photographs.

No series of pictures, however good, can replace practical experience gained in the examination of patients, but this atlas goes a long way to supplementing experience and filling in any gaps in knowledge of diseases of the ear, nose, and throat. Finally, it is unique in the number and high quality of its colour photographs. Any specialist will study it with pleasure and envy.

W. M. MOLLISON.

SIDE-EFFECTS OF CORTISONE

Untoward Reactions of Cortisone and A.C.T.H. By Vincent J. Derbes, M.D., F.A.C.P., and Thomas E. Weiss, M.D. (Pp. 51. 8s. 6d.) Oxford: Blackwell Scientific Publications. 1952.

Cortisone and A.C.T.H. are two of the most powerful and important drugs which have recently been made available. The work of Hench and more recent observers has made it clear that they may prove to be valuable therapeutic agents when their mode of action is better understood, but that in their present form their use presents a challenging problem—namely, the untoward reactions which may result from poorly directed, promiscuous, or prolonged administration. Most of the side-effects encountered are minor and reversible and are physiological rather than toxic in nature. But others are serious and may prove to be progressive, and even lead to loss of life. The authors of this small book record and briefly discuss the undesirable reactions which have been observed and recorded in the literature from the first use of these hormones in 1946 until December, 1951.

It seems doubtful whether an ephemeral record of this sort justifies publication in book form, particularly as most of the contents are now common knowledge among physicians with any experience in this field. But it is useful to have this concise tabulated account of the side-effects which have been observed during the past three years. Doubtless, as reports accumulate on the progress of series of cases subjected to long-term therapy, further complications will have to be recorded, while modifications of the substance themselves, or the emergence of efficient analogues, may be expected to alter the present position considerably. The side-effects are listed as they affect the various systems of the body, and there is a brief final chapter on treatment, together with what seems to be a fairly complete bibliography.

W. S. C. COPEMAN.

CONGRESS OF HAEMATOLOGY

Proceedings of the Third International Congress of the International Society of Hematology. Cambridge, England, August 21–25, 1950. Editor-in-Chief, Carl V Moore. (Pp. 593; illustrated. £3 10s.) New York: Grune and Stratton. London: William Heinemann. 1951.

This beautifully produced volume, which has a similar format to the journal *Blood*, is likely to appeal most to those who were actually present at the Congress of Haematology and wish to revive their memory of the subjects discussed there. It is conveniently divided into sections—anaemia and related subjects, immunohaematology, leukaemia and related diseases, coagulation, purpura and related subjects, and finally miscellaneous papers. Many papers are given in full, others in abstract, and others by title only. It is a pity that the editor-in-chief, Carl Moore, has been so diffident as to print only the title of his paper on the contribution of radioactive iron to the understanding of iron metabolism, which was one of the most appreciated papers at the Congress.

L. J. WITTS.

The fifth edition of Sir Lionel Whitby's *Medical Bacteriology* (London: J. and A. Churchill; 22s. 6d.), of which Dr. Martin Hynes is co-author, bears little resemblance to its predecessor. The number of pages describing the main bacterial genera has been almost trebled, and the "applied" section reduced, the alphabetical chapter of diseases with methods for their laboratory diagnosis disappearing altogether. This expansion of the central and most important part of the book has greatly improved it as a student's manual, and the remaining final chapters, including those on protozoa and helminths, are a useful addition.

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VALVOTOMY FOR MITRAL STENOSIS

Rapid progress has been made in the surgical treatment of mitral stenosis in the past two years since the first reports[1 2] were published showing that direct operation on the mitral valve was a reasonably safe procedure and that the majority of patients submitted to it were greatly improved. In the opening pages of this issue Mr. R. C. Brock and Drs. Charles Baker, Maurice Campbell, and Paul Wood report the results obtained in 100 patients: the progress of the first 50 to be operated on has now been followed for at least six months. In the whole series of 100 cases there were 13 deaths, seven occurring in the first 20 cases, so that in the last 80 cases the mortality was under 8%. These results become even more impressive when it is seen that 49 out of the first 50 cases were classified as being seriously or completely incapacitated by dyspnoea, orthopnoea, or pulmonary oedema. The effect of treatment is often spectacular. In the 41 surviving patients out of the first 50 operated on (in one case valvotomy was found not to be possible) the result is described as good or excellent in 32. Seventeen of these were restored to normal health and activity after having been completely incapacitated before operation. In a series of 352 cases collected from seven surgical centres Bland[3] found that three-quarters were improved, the mortality being 15%. Already, as the low mortality and good results of mitral valvotomy have become known to doctors and patients, there is great pressure on those surgical centres where this work is being carried on, and the waiting-lists are long. But not all patients with mitral stenosis are suitable for or in need of a valvotomy, and careful selection is essential. The disease is not necessarily progressive, and only a minority of those with little or no cardiac enlargement ever develop serious symptoms.[4 5] In symptomless mitral stenosis operation is unnecessary: it should be reserved for those patients who do develop symptoms at a later stage.

The selection of cases is largely influenced by the severity of the respiratory symptoms which are caused by pulmonary congestion. If, as seems certain, the operation of mitral valvotomy is to be more widely carried out the cause of the dyspnoea in each case must be critically assessed. Many patients with minimal valve damage are dyspnoeic either because of unwarranted limitation of their physical activities which has induced a cardiac neurosis or because of a complication such as obesity, pulmonary infection, or anaemia. Dyspnoea is a symptom which is notoriously difficult to assess, and in heart disease, as has already been shown in chronic pulmonary disease, any new and spectacular treatment is likely to cause temporary improvement. For this reason selection of patients for operation should be the responsibility of a physician working in close collaboration with the surgeon, so that only those patients whose disability can be shown to be largely due to the mechanical obstruction at the mitral valve will be operated on. Priority must be given to those with severe mitral stenosis but with little cardiac enlargement who have increasing dyspnoea and recurrent attacks of pulmonary oedema and haemoptysis. Many of these patients have died in an attack of pulmonary oedema while waiting for the operation which would have restored them to normal activity. Mitral valvotomy, by relieving pulmonary congestion and pulmonary hypertension, has in fact improved patients with congestive failure, greatly enlarged hearts, and presumably severe myocardial damage. Active rheumatism is at present considered to be an absolute contraindication to operation, and therefore valvotomy is rarely carried out until after the age of 20, when recrudescences of activity are less likely. Even when infection has been thought clinically to be inactive, biopsies of the auricular appendage at operation have shown Aschoff nodes in 4 of 11 specimens,[6] although in one other published case subsequent careful examination of the rest of the heart at necropsy failed to show any other signs of activity. Brock and his colleagues have shown that, even in the presence of aortic valvular disease, valvular calcification, or auricular fibrillation, good results can be obtained, though with a higher mortality.

The diagnosis of mitral incompetence in patients with mitral stenosis has proved to be unexpectedly difficult, and it now appears that the only sure way is to feel the regurgitant stream of blood with the finger in the left auricle. The loudness of the mitral systolic murmur often gives some indication of the extent of the incompetence, but many patients with loud murmurs are found at operation to have pure stenosis, while conversely the valve may be found to

[1] Baker, C., Brock, R. C., and Campbell, M., *British Medical Journal*, 1950, **1**, 1283.
[2] Glover, R. P., O'Neill, T. J. E., and Bailey, C. P., *Circulation*, 1950, **1**, 329.
[3] Ibid., 1952, **5**, 290.
[4] Grant, R. T., *Heart*, 1933, **16**, 275.
[5] Bland, E. F., and Jones, T. D., *Circulation*, 1951, **4**, 836.
[6] Kuschner, M., Ferrer, M. I., Harvey, R. M., and Wylie, R. H., *Amer. Heart J.*, 1952, **43**, 286.
[7] Gorlin, R., and Gorlin, S. G., ibid., 1951, **41**, 1.
[8] Lewis, B. M., Gorlin, R., Houssay, H. E. J., Haynes, F. W., and Dexter, L., ibid., 1952, **43**, 2.
[9] Munnell, E. R., and Lam, C. R., *Circulation*, 1951, **4**, 321.

be freely incompetent though clinically there was no suspicion of this. A large V wave in the pulmonary "capillary" pressure tracing coinciding with ventricular systole is often found in mitral incompetence, but the technical difficulties in recording accurate tracings renders this method of little practical importance in the diagnosis of incompetence. The presence of free mitral incompetence is a contraindication to valvotomy, and operation should not be carried out if there is gross enlargement of the left auricle with marked expansion during ventricular systole or if there is conspicuous left ventricular enlargement. A successful valvotomy, in addition to relieving the stenosis, increases the mobility of the valve cusps and may make valve closure more efficient; for this reason a lesser degree of mitral incompetence is no contraindication.

The success of mitral valvotomy in relieving the symptoms of mitral stenosis has led to a renewed interest in the changes in the pulmonary circulation which can be measured by the cardiac catheter. The pulmonary "capillary" pressure measured with the catheter tip impacted in a pulmonary arteriole gives an approximate estimate of the pulmonary venous and left auricular pressures, and by means of an ingenious formula the approximate size of the mitral valve opening can be calculated.[7] Lewis and his colleagues[8] have shown that with the normal valve area of 4 sq. cm. a large increase in flow through the mitral valve can result from only a slight rise in left auricular pressure. With moderate stenosis (valve opening of 2.5 sq. cm.) the resting pressure may be slightly increased, but on effort the pulmonary capillary pressure rises to a figure which may equal the plasma osmotic pressure. In severe stenosis, with a valve area less than 1 sq. cm.—most of the patients who are at present operated on come into this group—the resting pulmonary capillary pressure and the pulmonary arterial pressure are raised, the latter in some cases to over 100 mm. Hg. The post-operative course of these patients with marked pulmonary hypertension is of great interest. Structural changes in the pulmonary arterioles are commonly found in severe mitral stenosis, and it seemed likely that these changes would persist after a successful valvotomy which has relieved the pulmonary capillary congestion. Direct measurement at operation has shown that the raised left auricular pressure returns to normal and very high pulmonary artery pressures may be reduced immediately by over 50%.[9] Although in the preoperative state the high pulmonary arteriolar resistance may be a compensatory mechanism protecting the pulmonary capillaries from a high pulmonary artery pressure which would otherwise cause pulmonary oedema, its persistence after operation might have an adverse effect on the patient's later progress. Catheterization of Mr. Brock's patients many months after operation confirms the significant lowering of pulmonary arterial pressure, and it is gratifying to find that even severe pulmonary hypertension in mitral stenosis is reversible.

A much longer period of observation will be needed to see how permanent are the results of mitral valvotomy, for although the obstruction at the mitral valve is relieved and the pulmonary hypertension is usually reversible the damage to the heart muscle or further scarring of the valve may cause a return of symptoms. However, it is already apparent that a great advance in the treatment of rheumatic heart disease has occurred and that a large number of patients at present seriously incapacitated can be enabled to live normal lives.

FUTURE FOR OPTICIANS

Before the *Report of the Interdepartmental Committee on the Statutory Registration of Opticians*[1] produced under Lord Crook's chairmanship can be seen in perspective, certain features of its background must be clarified. Many medical men have maintained that, although retinoscopy and other objective refraction tests can be learned in a comparatively short time, the capacity to interpret subjective tests cannot be acquired without a full medical training. They have also been at pains to emphasize that the human eye, linked as it is with the body and mind as a whole, must suffer if its examination is entrusted to opticians, because opticians are necessarily ignorant of medicine. Furthermore, they have collected instances of allegedly unnecessary glasses prescribed by opticians, together with examples of patients in whom an optician has failed to detect serious eye disease. In reply to these criticisms it has been urged by opticians (*a*) that they in their turn can point to errors of omission and commission by ophthalmologists; (*b*) that opticians must inevitably do most of the nation's refraction work, because there are not nearly enough trained medical men to cope with it; and (*c*) that they are well aware of their medical ignorance, but long to remedy that defect through instruction given by medical men. This latter plea has hitherto been resisted by a strong section of doctors, mainly on the ground that unscrupulous opticians, having once gained a smattering of medicine, could walk out of their fostering hospital and proceed to exploit their new knowledge by advertising. To this objection representatives of the optical

[1] H.M.S.O., Cmd. 8531. Price 1s. 3d.

profession have answered that they themselves have discouraged advertisement, and that they are eager to associate themselves with doctors in the setting-up of a disciplinary body empowered to stamp out advertising and all other unprofessional activities. Such a disciplinary body could also regulate the standards of optical education, and would obviously entail an official register.

In the light of these considerations it is significant that the establishment by legislation of a General Optical Council, analogous to the General Medical Council, is the first of the Crook Committee's recommendations. This new Council, sitting under an independent chairman, would include two nominees of the Privy Council and seventeen other members representing the optical examining bodies, the ophthalmic opticians, the dispensing opticians, and the medical profession. Probably there would be a registrar, appointed in the first instance by the Privy Council, and thereafter by the General Optical Council itself. One of the five medical representatives should in the Crook Committee's opinion be a general practitioner. This is assuredly a wise provision which will be approved by the general public as well as by the medical profession. A statesmanlike family doctor, loyal to his own colleagues but capable of seeing the opticians' point of view, familiar with the problems of everyday practice, and vigilant in all matters pertaining to the welfare of patients, might well become the most valued member of the Council. Clearly the initial composition of such a Council will be of vital importance, because "all the whetting in the world can never set a razor's edge on that which hath no steel in it." A mass of important detail remains to be worked out by the Council, which will at once be confronted by pressing questions, such as the proper scope of the optician in orthoptics and contact lens fitting. Regulations will have to be prepared concerning the use of drugs by opticians, and safeguards must be devised to satisfy ophthalmologists who would not otherwise agree to clinical teaching for opticians.

The report suggests that ophthalmic opticians who are statutorily registered should be recognized as competent to test vision and to determine and provide any necessary optical correction. The report goes on to say (p. 28, para. 118): "They must, of course, have knowledge of ocular abnormalities, as they may in the course of their professional duties suspect the presence of disease. But their registration should not imply an ability to diagnose ocular or other diseases. They would continue, wherever they suspect a departure from the normal, to refer their patients for medical advice." These sentences should help to allay the suspicions of all those medical men who are not irreconcilably averse to clinical teaching for opticians, especially if they bear in mind that the General Optical Council, as envisaged by the Crook Committee, will have the power to punish any abuse of such teaching. The desirability of clinical instruction by medical men is more than once stressed in the report (see particularly p. 26, para. 104), though the authors realize that most of this instruction might have to be done in hospitals not already committed to a large programme of teaching for undergraduate and postgraduate medical students. Amicable arrangements for the clinical instruction of opticians might fix the seal of good will between oculist and optician.

At least an attempt should be made to approach the matter in an unbiased fashion, and both sides should cease to harbour dark suspicions of each other. While the profession will be wise to examine the Crook Report with a close scrutiny, and not hesitate to be critical, we must consider whether there is any decent and feasible alternative to the friendly co-operation therein advocated between medical men and opticians. There are not enough doctors to do all the people's eye examinations—not even if the number of doctors trained in ophthalmology could be increased threefold overnight. It is equally unthinkable that opticians isolated from contact with medicine could be allotted sole responsibility for the work. Difficulties lie ahead, but the difficulties, as Mr. Churchill remarked in another context, will speak for themselves. We need not mention them now, except to say that they can be surmounted if oculist and optician will respond to the spirit of the Crook Report. This document is full of tolerance and common sense. It does not seek to hide the fact that some of the evidence which its authors had to sift was conflicting, but its keynotes are restrained optimism and a genuine concern to help.

A NEW ANTIMALARIAL DRUG

While testing a range of compounds for folic acid antagonism, Hitchings and his colleagues[1] were struck by a similarity in the behaviour of proguanil ("paludrine") and certain pyrimidines. This observation prompted a further comparison of the properties of these compounds, and it at once became obvious that, weight for weight, many of the pyrimidines were more powerful antimalarial drugs than proguanil. They then synthesized and tested a large number of pyrimidine derivatives,[2] and the most active of these was selected for further trials. This is 2:4-diamino-5-(4-chlorophenyl)-6-ethyl pyrimidine, first referred to as B.W. 50–63, and now known as "daraprim." Compared with proguanil daraprim is some 60 times as active against *Plasmodium gallinaceum* in the chick and 200 times as active against *P. berghei* in the mouse. In low concentrations it clears *P. cynomolgi* from the peripheral circu-

lation of the monkey and *P. cathemerium* from the canary. It has been found by Schneider, Montezin, and Biheu[3] to have marked activity against *P. relictum* in canaries. Little has been published about the development of strains of the parasite resistant to daraprim. Some observations by Rollo[4] suggest that this drug is less likely to produce such strains than is proguanil, but until more extensive and more detailed work has been completed it would be well to maintain an attitude of caution.

Toxicity tests in rats and mice have been satisfactory, the LD50 being less than that of proguanil, while no symptoms suggesting chronic toxicity have arisen after prolonged administration. A group of human volunteers took 50 mg. twice weekly over a period of three months without untoward results, and Goodwin[5] took varying doses of the drug continuously for a year. In none of the clinical trials have unpleasant side-effects been noted. The first therapeutic trials were carried out in Lagos by Archibald[6] in symptomless infections in schoolchildren. He found that a single dose of 5 mg. was sufficient to clear the blood stream of the asexual parasites of *P. falciparum* and *P. malariae* for periods of about 30 days, though gametocytes of the former species persisted for a long time. McGregor and Dean Smith,[7] working in Gambia, found that a single dose of 0.25 or 0.5 mg./kg., given to infants suffering from acute malaria, cured the fever and rendered the blood smear negative. The infants remained well for some weeks, but because there was constant exposure to reinfection it was impossible to determine whether or not cure was complete. One patient in this series failed to respond to daraprim, but the authors suggest that this may have been related to a concurrent hepatitis from which the child suffered. It is to be noted that in both these trials parasites and fever were slow to disappear, a point which some consider a disadvantage. Unlike most other antimalarial drugs, daraprim is tasteless and can be given in the form of a syrup to infants.

Schneider, Canet, and Dupoux,[8] who gave the drug in various doses, have carried out a more comprehensive series of trials in Tunisia and Indo-China. They advocate a single dose of 50 mg., or at most two doses of 50 mg. on successive days. Their results are similar to those of the West African workers, except that they found the drug to act more rapidly and in this respect consider that it compares favourably with chloroquine ("nivaquine"). The majority of their patients were infected with *P. falciparum*, but some, who reacted equally well if more slowly, were infected with *P. vivax*. Vincke[9] has recently reported from the Belgian Congo the results of an experiment in mass treatment. The inhabitants of two isolated villages were given a weekly dose of 25 mg. for eleven weeks during the season of transmission (September to December). At the beginning of the experiment the parasite index was approximately 22%, at the end 1.3%, and a month later blood tests on all the subjects were negative, despite the fact that many failed to take the full 11 doses. The sporozoite index in the mosquitoes was reduced to a very low figure, though transmission was not completely broken, probably because of the presence of infected strangers visiting or passing through the area. At the time of year when the experiment was carried out the sporozoite index usually rises steeply. The only published experiment in suppression or prophylaxis is that reported in this *Journal* by Goodwin,[5] who, while taking daily doses of 5 mg. of daraprim, allowed himself to be bitten by four mosquitoes known, and afterwards proved by dissection, to carry sporozoites of *P. falciparum*. It is now over nine months since this experiment, and infection has not developed.

These few trials, which are all that have been reported to date, are too limited in their scope to allow a definite opinion to be expressed on the merits or shortcomings of daraprim, but they show clearly that it is a very potent antimalarial drug. Its most important use may eventually be as a suppressant. Points in its favour are its tastelessness, the complete absence of side-effects, and the fact that it may prove to be effective in weekly doses. The results of further trials will be awaited with interest.

1 *Nature, Lond.*, 1949, **164**, 107.
2 *Brit. J. Pharmacol.*, 1951, **6**, 185.
3 *Bull. Soc. Path. exot.*, 1952, **45**, 29.
4 *Nature, Lond.*, 1951, **168**, 332.
5 *British Medical Journal*, 1952, **1**, 732.
6 Ibid., 1951, **2**, 821.
7 Ibid., 1952, **1**, 730.
8 *Bull. Soc. Path. exot.*, 1952, **45**, 33.
9 *Ann. Soc. belge Méd. trop.*, 1952, **32**, 91.

STREPTOMYCIN AND DIHYDROSTREPTOMYCIN

The introduction of *iso*nicotinic acid hydrazide has not made streptomycin obsolete in the treatment of tuberculosis. It will be many months before the potential value of the new drug can be determined, and even then streptomycin may well remain the foundation of the antibacterial treatment of the disease. Consideration of the toxic effects of streptomycin and dihydrostreptomycin on the 8th cranial nerve is therefore still topical and important. The subject was recently discussed at a meeting of the Otology Section of the Royal Society of Medicine. Although the toxic effects of both these antibiotics were fully described, little was said about the important practical question of which should be used in treatment. If one were more effective than the other there would be no problem: but there appears to be little, if any, difference between them in this respect.[1] The choice therefore must depend entirely on their toxicity. It is now considered that a dose of 1 g. of streptomycin daily is adequate in most varieties of tuberculous disease, and there is some evidence that in certain types of pulmonary tuberculosis this dose need be given only

1 Council on Pharmacy and Chemistry, *J. Amer. med. Ass.*, 1951, **147**, 253.
2 Tempel, C. W., Hughes, F. J., Mardis, R. E., Towbin, M. N., and Dye, W. E., *Amer. Rev. Tuberc.*, 1951, **63**, 295.
3 Biagin, R. W., *British Medical Journal*, 1951, **2**, 651.
4 Cathie, I. A. B., and Garrow, D. H., ibid., 1951, **2**, 735.
5 Somner, A. R., ibid., 1951, **2**, 970.
6 Grant, J., ibid., 1951, **2**, 1221.
7 Sher, N., ibid., 1951, **2**, 1521.
8 Naismith, J. T., ibid., 1952, **1**, 796.
9 Glorig, A., Romansky, M., and Katz, S., *Trans. 10th Conf. Chemo. of Tuberc., Vet. Admin.*, 1951, p. 227.
10 O'Connor, J. B., Christie, F. J., and Howlett, K. S., *Amer. Rev. Tuberc.*, 1951, **63**, 312.
11 Don, C., and Gregory, J., *Lancet*, 1952, **1**, 72.
12 Canada, R. O., Allison, S. T., D'Esopo, N. D., Dunner, E., Moyer, R. E., Shamaskin, A., Tempel, C. W., and Charter, W. V., *Amer. Rev. Tuberc.*, 1950, **62**, 563.
13 Bignall, J. R., Crofton, J. W., and Thomas, J. A. B., *British Medical Journal*, 1952, **1**, 554.

every third day.[2] With these doses the differences between the incidence and severity of the vestibular damage caused by streptomycin and dihydrostreptomycin are very small and of no practical significance. But the drugs differ much more in their effects on the cochlea. There have been many reports of an increased incidence of deafness with dihydrostreptomycin used for tuberculous meningitis.[3-8] Professor F. C. Ormerod and his colleagues at the Institute of Laryngology clearly demonstrated at the meeting that there may be considerable tuberculous infiltration of the cochlear portion of the 8th nerve of patients with meningitis who have become deaf during streptomycin treatment. It is impossible to determine during life whether the deafness is due to the disease or the treatment. The study of patients with tuberculous meningitis is therefore not a satisfactory way of comparing the toxicity of the drugs. In those with pulmonary disease the cause of the deafness is less doubtful, and comparison is easier since even slight cochlear damage very rarely results from streptomycin treatment in daily doses of 1 g. With dihydrostreptomycin severe damage has been reported in several patients in the United States of America and in this country.[9-11] There has been some discussion whether it is due to the dihydrostreptomycin or to an impurity. But the proof that an impurity is the cause would not alter the fact that the commercial product at present available can make a patient deaf. Compensation for the labyrinthine damage usually takes place, and the risk of permanent disability, except in the elderly, is very small. There may in addition be recovery of labyrinthine function, as shown by caloric tests.[12 13] But so far as is known at present the cochlear damage is irreversible, and there can of course be no compensation for it. The exact incidence of deafness with different makes and batches of dihydrostreptomycin is of little importance. If there is a choice between two drugs of equal therapeutic effect, one liable to cause temporary inconvenience and the other permanent disability, it is surely irrelevant to contrast how many will be inconvenienced with how few will be disabled. Except in the very rare instances of severe sensitivity reactions to streptomycin in which desensitization has failed, there seems to be no reason why dihydrostreptomycin should be used at all in the treatment of tuberculosis.

THE WORK OF HEALTH VISITORS

In his last annual report[1] to the County Council of the West Riding of Yorkshire Professor C. Fraser Brockington discusses one of the difficulties of the Welfare State—the multiplicity of social workers employed by different departments. He writes:

"Yet the problems of family life cannot be separated into watertight compartments so that Miss A can visit to deal with the problems of baby care, Miss B with little Tommy who has been found at school to have some defect, Miss C with the foster child, Miss D with the elder boy who is under the care of the mental health committee, and Miss E with the aged grandfather now beginning to fail. The social problems involved in all these, and other similar situations, are related one to another in such a way that they can rarely be dealt with separately."

Professor Brockington goes on to recommend the employment of "a common basic social worker" throughout the whole field of socio-medical work, and he thinks the person most fitted for such work is the health visitor. This is a logical suggestion, and it would probably be fairly easy to put it into effect in rural areas if not in thickly populated towns. But apart from anything else the number of trained health visitors is still too few to fill all the vacancies, and of course existing legislation—for instance, the Children Act—does not permit health visitors to carry out some of the functions mentioned by Professor Brockington. Again, there will always be a need for specialist social workers such as probation officers, psychiatric social workers, and hospital almoners. The World Health Organization is at present investigating the duties of social workers in this and other European countries, and no doubt the question of overlapping will be one of the points to be considered.

When the N.H.S. Act came into force it looked for a time as if there might be some duplication of home visiting by almoners and health visitors, but in many areas of the country there is now excellent opportunity for almoners and health visitors to work as a team for the benefit of patients discharged from hospital as well as of those attending as out-patients.

The official view of a health visitor's duties was outlined in a pamphlet[2] prepared not long ago by the Ministry of Health. In this she is described as a nurse with special training in health education, child welfare, and social work who

"hitherto has specialized mainly in mothercraft, though some health visitors have visited the homes of sufferers from tuberculosis, mental illness, and venereal disease. But under the new Service she is the friend and adviser of the whole family . . . the aim is that she should work in much closer contact with the family doctor, so that he can readily call her in when he finds a family needing advice or social service which she can give or arrange."

For a long time many general practitioners refused to welcome the appearance of health visitors in their districts, fearing with some justification that clashes of opinion would be bound to occur. These fears have largely subsided, but medical officers of health are still doubtful whether general practitioners are ready to accept the assistance of health visitors in the way envisaged by the Act. In fact it is not known how urgently general practitioners feel a need for the sort of help which health visitors might be able to give. The experimental health centres which are being established with the help of the Nuffield Provincial Hospitals Trust[3] will in due course provide useful information about these problems of socio-medical work.

The next session of the General Medical Council will open on Tuesday, May 27, at 2 p.m., when the President, Professor David Campbell, will take the chair and will deliver an address.

[1] Sixty-second Annual Report (1950) of the County Medical Officer of the County Council of the West Riding of Yorkshire.
[2] *The National Health Service*, 1949, H.M.S.O., London.
[3] Report of the Nuffield Provincial Hospitals Trust, 1948–51. See *British Medical Journal*, 1951, **2**, 1326.

REFRESHER COURSE FOR GENERAL PRACTITIONERS

TREATMENT OF BURNS—II

BY

J. P. BULL, M.D. AND D. M. JACKSON, M.D., F.R.C.S.

(From the M.R.C. Burns Unit, Birmingham Accident Hospital)

A further important step in the prevention of infection in burns is early grafting. Only when a wound has healed is the risk of infection finally over. For this reason, as well as to minimize scarring, skin grafts are regarded as the best dressing for a granulating area and should be used as early as possible.

Depth of Burning

The modern classification of the depth of burning is into two degrees—partial-thickness and full-thickness skin-loss. Every recent burn which is diagnosed as full-thickness skin-loss should be considered for excision down to normal tissue followed by immediate grafting. Sometimes this is contraindicated by shock, or by difficulty in diagnosing full-thickness skin-loss or in defining its limits, but excision remains the ideal treatment. If, however, this treatment is not possible, the area of complete skin destruction can always be diagnosed by the end of three weeks, and usually by 16 days after injury, and grafting should not be delayed beyond this time. Every full-thickness burn should therefore be grafted either at once or at the latest within three weeks of injury.

The diagnosis of the depth of a burn is notoriously difficult. Whether partial- or full-thickness skin-loss is present depends on whether the bases of the hair follicles and sweat ducts are destroyed, and this cannot be discovered by inspection of the surface of the skin. A white avascular coagulated surface is no indication of a full-thickness skin-loss burn; in fact, on the face, scalp, palms, and soles it often heals as partial skin-loss within a fortnight. Similarly, within 24 hours of the injury a visible circulation in the burned skin, as judged by redness which blanches on pressure, is no certain indication of partial-thickness loss; for circulation, initially present, may cease several hours after burning, and the lesion may then prove to be full-thickness skin-loss.

The pin-prick test for analgesia is a useful and quick guide to the depth of burning. If part of a burn is pricked repeatedly and firmly with a sterile hypodermic needle, and some pain sensation is present, that part of the burn will heal as partial-thickness skin-loss within three weeks. If analgesia is present (and the burn is not produced by a chemical such as carbolic acid), it is either full-thickness loss or very deep partial skin-loss, taking over three weeks to heal. In certain sites where the skin is thin, such as the back of the hand and fingers and the dorsum of the foot, this test is often enough to indicate primary excision and grafting; but on the face, scalp, palms, and soles the epithelial elements extend more deeply and analgesia is by no means sufficient evidence for primary excision. Although this test is easy to perform, it must be interpreted carefully in conjunction with the history and the site of burning if the excision of partial-thickness skin-loss burns is to be avoided. Generally speaking any area of full-thickness skin-loss more than 1 inch (2.5 cm.) across at its narrowest diameter will take three weeks or more to heal, and should be grafted at once. On the face and fingers even smaller areas may best be treated by grafting.

Grafting of Full-thickness Burns

The most useful type of graft for resurfacing burns is the split-skin graft cut with a Blair knife. The superficial quarter or half of the thickness of the skin is taken in this way (see Diagram) and a donor area is left which heals in 10 days and can be used to supply further grafts in 16 to 20 days. This is the type of graft used routinely on excised and granulating areas and where the texture of the skin is unimportant: in areas where thicker skin of uniform texture is required, and where avoidance of graft contraction is important, a three-quarter-thickness dermatome graft, a full-thickness graft, or a skin flap should be used. One of the advantages of primary excision is that a full-thickness graft, if indicated, can be applied initially rather than at a second operation.

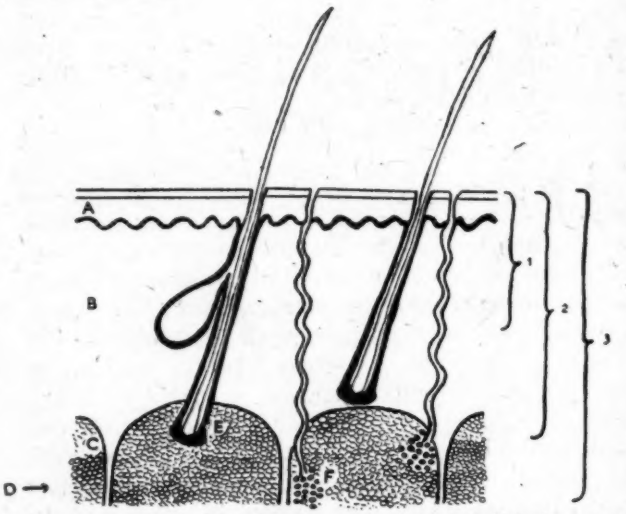

Diagram of the skin showing the position of epithelial elements. A=Epidermis. B=Dermis. C=Subcutaneous tissue. D=The plane dividing partial-thickness from full-thickness skin-loss. E=Hair follicle. F=Sweat gland and duct. 1=Thin or intermediate split-skin graft (usually cut with a Blair knife). 2=Three-quarter-thickness split-skin graft (usually cut with a dermatome). 3=Full-thickness skin graft.

Extensive Burns

The management of extensive full-thickness skin-loss burns raises further problems. Frequently the first of these is how to keep the patient alive until grafting is possible two and a half weeks after injury. Blood transfusion and artificial feeding are important aids to this end.

The haemoglobin percentage should never be allowed to drop below 80%, and between 120% and 130% is the figure at which to aim when estimating the amount of blood to be transfused. Recently a patient with a 64% burn who survived required 20 litres of blood between injury and complete healing: this is an unusual amount, but it illustrates the large transfusions sometimes needed. When repeated transfusions are likely it is important that blood should be of the patient's own group and Rh factor, and the greatest economy in veins is called for. Haemoglobin estimations should be carried out every two or three days in these cases, as a fall of 5–10% daily may occur.

After the stage of shock anorexia is common and difficulty is often experienced in getting the extensively burned patient to take food. If enough protein can be given to patients in this condition, the appetite is usually restored and they begin to take ordinary meals once more. Usually, however, an ordinary full diet is inadequate to prevent gross

wasting due to the utilization of endogenous protein. A high protein and high calorie diet is required, and this may conveniently be given as a continuous drip feed through an indwelling nasal tube. A useful mixture is: 18 oz. (511 ml.) milk, two eggs, 1½ oz. (42 g.) milk protein, and 1 oz. (28 g.) sugar. This provides 780 calories to the pint (568 ml.), and the usual supplementary feed is 3 to 4 pints (about 2 litres) a day. This type of supplementary feeding should probably be given from the second day to all burned patients who have sustained more than about 20% of full-thickness skin-loss.

A further problem is how to secure skin cover as soon as extensive areas of complete skin destruction begin to granulate. Skin grafting in these cases is an urgent matter, and many patients have died because the danger of delay was not realized by the surgeon. A few of the principles of grafting extensive areas may be mentioned.

1. The maximum amount of skin cover should be obtained at the first operation; the importance of this is shown by the dramatic improvement which often follows during the 24 hours after a large burn is covered.

2. Great care must be taken in preparing patients for extensive grafting operations. The haemoglobin must be at least 80%, and a blood transfusion must be running before an area of 5% or more of full-thickness skin-loss is prepared and grafted. In suitable cases tourniquets, elevation of the limbs, and tilting the operating table can be used to limit blood loss.

3. If the whole burn cannot be grafted at once, the critical areas must be done first; they are the face and neck, hands and feet, and the flexor surfaces of joints.

4. The grafting programme should be planned so that the best use can be made of the donor areas. Where it is avoidable the patient should not be turned over while under the anaesthetic. Even with careful planning it is easy for the surgeon to become unpunctual in carrying out his own grafting time-table.

5. An early decision should be made on the suitability of a case for the use of skin grafts from another person (homografts). If homografts are indicated they should be used early in treatment to gain the greatest benefit from skin cover. When applied in sheets homografts take like autografts, but disintegrate about three weeks later after an immunity has developed against them, leaving the area granulating once more. By using ¼-in. (1.3-cm.) wide strips of homograft, and alternating these with similar strips of autograft, the homografts *appear* to take permanently. It is probable, however, that the epithelial part of the homograft is replaced by autograft epithelium before immunity has developed.

6. All unused skin graft should be stored in a refrigerator: it will then remain viable up to three weeks, and probably up to six weeks. Small areas of graft failure can be dressed with this stored skin and further operation so avoided.

Special Features of Burns of Certain Sites

Hands.—In most full-thickness skin-loss burns of the hand in civilian practice only the skin and subcutaneous tissue are destroyed; in these cases early full function can be obtained, even if the whole hand is granulating two and a half weeks after the accident. The essential features of treatment to obtain this result are elevation of the limb from the first day to control oedema, immobilization of the hand and wrist in the position of function until epithelization is complete, and the completion of skin cover within three to four weeks of the injury. Sheets of split-skin graft are satisfactory for the back of the fingers and hand, but on the palmar surface a three-quarter-thickness dermatome graft after primary excision gives a better result. Following flexor surface grafts of the fingers splinting in extension is advisable for two to three months, and this can be maintained conveniently without complete immobilization by using a leather glove with stay bones incorporated over the backs of the fingers, hand, and wrist. Deep lesions involving tendons, nerves, vessels, and bones are usually the result of burns during unconsciousness, and burns by electricity or by the hand being firmly held between hot rollers or in faradic spasm on an electric fire bar. Immediate excision of these deep burns is often unwise, since it is exceedingly difficult to define the limits of necrosis in the first few days. Nevertheless, excision of non-viable tissue is still indicated as soon as demarcation is present, and this is essential if healing is to be complete within the critical three to four weeks. Surgery in these cases has the same objective as in other types of hand injury—to restore the action of the hand as a pincer and a vice before it can become stiff and useless.

Face.—It is generally recognized that full-thickness skin-loss burns of the eyelids should be grafted as soon as possible to prevent ectropion, which may possibly lead to corneal ulceration and blindness. It is important, however, that the whole face and neck should be treated as a critical area and grafted not later than three weeks. Areas of complete skin destruction on the head and neck often result from falling into the fire in an epileptic fit. These burns can usually be excised and replaced with a dermatome graft on the day of injury, and the cosmetic result may be so good that further treatment is unnecessary.

Feet.—It is not generally appreciated that split-skin grafts cut with a Blair knife give excellent results in deep burns of the feet. Even on the sole and over the tendo Achillis they stand up well to rough wear, and they have proved satisfactory as the routine graft for molten-metal burns in foundry workers, who stand for long hours in heavy boots in a hot, dirty environment. They are contraindicated only if the burn has destroyed the pad of subcutaneous tissue or paratenon, and bone, joint, tendon, or nerves are exposed. After grafts on the feet have taken, controlled return to walking is necessary to prevent damage by capillary haemorrhages beneath the graft. This danger is avoided by dressing the graft on the fifth day after operation, and, if it has taken satisfactorily, giving active exercises in bed for two days, hanging the legs over the edge of the bed for one day, sitting out for one day, and walking on the tenth day.

Bone.—A free graft will take satisfactorily on periosteum, but, if the periosteum is destroyed by burning, the exposed bone should be regarded as dead. Burns down to and including bone, which occur most commonly on the skull, should be excised down to viable tissue and skin grafted to secure early healing. A split-skin graft on marrow is satisfactory for wound closure and sometimes as permanent skin cover for the skull, but it is not satisfactory where it will be subject to pressure or rubbing.

Joints.—In applying free grafts near to a joint there are several important principles which should be followed. The joint should be extended to make the burn on the flexor surface as large as possible before grafting. Scars between the graft and sound skin, and between separate sheets of graft, should be planned to run round the limb and not across the flexor crease, where they are likely to form tension bands. Splinting is never required for grafts on extensor surfaces if they are stretched from the start by active exercises. Often, however, grafts on flexor surfaces benefit from splinting combined with active exercises for periods up to three months. By this time the tendency for the graft to contract has usually ceased unless there is gross shortage of skin, when further grafting will be required.

Respiratory Passages.—It is unusual for the respiratory passages to be burned even when the face is severely burned by flames, presumably because the breath is held. A conflagration in an enclosed space is the commonest cause of this lesion; more rarely it may follow the swallowing of hot liquids. If the lungs are involved a large amount of exudate is rapidly poured out and the patient tends to drown. The prognosis is very grave and care should be taken not to give excessive amounts of intravenous fluid. Early tracheotomy, with administration of oxygen and frequent suction to keep the air passages clear, offers the best hope. Tracheotomy is also indicated for oedema of the glottis following the drinking of hot liquids if obstruction is severe. It is contraindicated for the noisy breathing and gross oedema of the subcutaneous tissue which accompanies burns of the neck; in these cases, if the patient sits erect in bed the oedema quickly descends on to the trunk.

Chemical and Electric Burns

Chemical Burns.—It is often impossible to tell at once whether a chemical burn from an unknown caustic is due to acid or alkali, so that valuable time may be wasted in searching for an appropriate antidote. Treatment is made easier by a buffer solution which will neutralize both types of caustic. A mixture of monobasic and dibasic phosphate is recommended which has the following composition: monobasic potassium phosphate (KH_2PO_4), 70 g., and dibasic sodium phosphate ($Na_2HPO_4.12H_2O$), 180 g., in 850 ml. of water; this makes 1,000 ml. of solution. The solution is non-irritant and can be used in the eyes. The caustic should be washed off immediately and vigorously either with water or with this solution and a compress of the buffer applied for 20 minutes. The chemical burn should then be treated as described for a thermal injury.

Electric Contact Burns.—These are usually painless and deep. Until the blistered skin is removed they tend to look unimportant, but because of their depth the slough is slow to separate and the scarring great. They can often be treated by primary excision and grafting, but it is sometimes difficult to decide how much tissue is devitalized. Electric contact burns on the hands may coagulate the tissues down to tendon, and in these circumstances a skin flap is indicated to preserve finger movement.

Conclusion

The modern management of burns owes much to new knowledge of physiology, bacteriology, and pathology. This has been combined with the general surgical principles of the treatment of open wounds and the special skills of plastic surgery. As a result many burned patients can now be saved whose injuries until recently would have been thought certainly fatal. For instance, instead of children with burns of one-third of the body surface probably dying, four-fifths of such patients should now survive. It is evident that further advances will be made. Much effort is now being applied to the prevention of burns and scalds, particularly of the all too frequent domestic injuries. The fuller understanding and control of the shock period, better prevention of infection, and technical improvements in grafting are all immediate possibilities. Assessment of the value of changes in detail of therapy is not easy and usually demands study of carefully controlled series. Comparison of mortality results has now been simplified so that series treated under different regimes can be validly compared from this point of view. The other two important criteria of success—speed of healing and completeness of function—should also be available when judging the value of new methods.

Next Refresher Course Article.—"Diseases of the Gall-bladder," by Mr. Harold C. Edwards.

The Minister of National Insurance has asked the National Insurance Advisory Committee to review, in the light of changes in the cost of living and other relevant circumstances, the amounts by which the National Insurance benefits of long-term hospital in-patients are reduced. Benefit is at present reduced after eight weeks in hospital by 5s. a week if the patient has a dependant, and by 10s. a week in other cases. After a year in hospital benefit is further adjusted, leaving the patient with 10s. a week if the treatment is for respiratory tuberculosis and with 5s. a week in other cases. But if the patient has a dependant the balance may be paid to the dependant. The committee will consider representations from interested organizations or persons. Representations, in writing, should be sent to the secretary, National Insurance Advisory Committee, 30, Euston Square, London, N.W.1, before June 20.

THE NEW MINISTER OF HEALTH

On May 7 it was announced from 10, Downing Street, that the Minister of Health, Captain H. F. C. Crookshank, had tendered his resignation, and that the Queen had been pleased to approve the appointment of Mr. I. N. Macleod as Minister of Health. Captain Crookshank succeeds Lord Salisbury as Lord Privy Seal and will continue his duties as Leader of the House of Commons.

The Rt. Hon. IAIN NORMAN MACLEOD, M.P., P.C., is 38 and the eldest son of a doctor, the late Dr. Norman Macleod, who for many years was in practice at Skipton, in Yorkshire. He was educated at Fettes and at Gonville and Caius College, Cambridge, where he took an honours degree in history.

[*Luther-Smith, Enfield*

In the 1945 General Election Mr. Macleod contested the Western Isles, a constituency in which he had previously lived but where no Conservative candidate had come forward since 1931; he was unsuccessful in a three-cornered fight. He returned to the Army and served for a while in Norway, before being demobilized in 1946.

After the war Mr. Macleod took to politics in good earnest. He joined the Conservative Parliamentary Secretariat, and worked under Mr. R. A. Butler. In 1948, when the Party's Home Affairs Research Department was established, he became its joint head with Brigadier Enoch Powell, and when the latter left he continued in sole charge until just before the 1950 General Election. This department provides background information for Conservative Members of Parliament, and also undertakes the detailed examination of policy. In 1946 Mr. Macleod was adopted as the prospective Conservative candidate for Enfield, Middlesex, and, after the division of the constituency under the Representation of the People Act, 1948, for Enfield West. He won this seat for the Conservatives in 1950 with a majority of over 9,000 in a three-cornered contest, a figure he improved upon in the 1951 election in a straight fight. After serving as secretary of his party's Parliamentary Committee on Insurance and Pensions in the 1950 Parliament, in the next Parliament Mr. Macleod was made chairman of the Conservative Parliamentary Committee on Health and Social Security in succession to Sir Hugh Lucas-Tooth. He was also chairman of a group of nine newly elected Conservative M.P.s who in 1950 published their views on home policy under the title *One Nation*. They dealt with such questions as the social effects of redistribution of wealth, population policy, housing, education, health, insurance and assistance, the care of old people, social policies for industry and the countryside, local government, and payment for the social services. Mr. Butler lent his weight to the book with a commendatory foreword. Mr. Macleod is joint author (with Brigadier Powell) of another political pamphlet, *The Social Services: Needs and Means*, an essay originating during the passage of the National Health Service and National Insurance Bills of 1951, when it appeared to the authors that neither major party had clearly thought out the relationship between the social services and the means test. This pamphlet was the subject of a recent leading article in the *Journal* (January 26, p. 207).

Since being appointed Minister of Health Mr. Macleod has been made a Privy Councillor. He does not have a seat in the Cabinet.

Correspondence

Because of the present high cost of producing the Journal, and the great pressure on our space, correspondents are asked to keep their letters short.

New Results with Isonicotinic Acid Hydrazide

SIR,—There is a danger that the indiscriminate use of *iso*-nicotinic acid hydrazide (I.N.H.) and its derivatives in the treatment of tuberculosis may have serious consequences. For this reason we should like to draw your attention at this stage to the following results which we and our colleagues have obtained in the last two months.

Cultural Tests.—(1) We have confirmed the American statement that the human tubercle bacillus (H37Rv) is sensitive to I.N.H. in concentrations of the order of 0.01 μg./ml. (1 part in 100 million). (2) Except perhaps in high concentrations the drug does not sterilize the cultures. The "sensitivity" depends on the period of incubation. In Dubos' liquid medium growth occurs in progressively higher concentrations of the drug, as incubation continues without subculture, until after three or four weeks' incubation the organisms have grown in as much as 8 μg./ml. (3) This increased tolerance of the drug can be delayed or abolished in the presence of streptomycin. (4) Resistance of human tubercle bacilli to I.N.H. can be rapidly increased by subculture in increasing drug concentrations. The strain of human tubercle bacillus is now growing, after only three subcultures, in as much as 62.5 μg. I.N.H. per ml.

This increase in resistance is more rapid, and therefore possibly more serious, than any increase described in the literature to which we have so far had access, including the *Quarterly Progress Report* of the Veterans Administration Study on the Chemotherapy of Tuberculosis, reviewed in your annotation last week (May 10, p. 1015).

Experimental Tuberculosis.—Results that have so far come to our notice suggest that animals previously subjected to an experimental infection of tuberculosis (bovine or human) were not only adequately protected from the disease by I.N.H., but also, when examined subsequently post mortem, "were free of viable tubercle bacilli" (Grunberg, E., and Schnitzer, R. J., *Quart. Bull. Sea View Hosp.*, 1952, **13**, 1). Detailed information on what occurs after therapy has been discontinued is unfortunately lacking.

Our provisional results with experimentally induced intracorneal tuberculosis in mice (a technique already shown to be valuable as an *in vivo* screening test for anti-tuberculous substances by Rees and Robson in *Brit. J. Pharmacol.*, 1950, 5, 77) may therefore be of interest. In such animals, I.N.H. administered in the diet at a dosage approximating to 12 mg. per kilogram body weight per day apparently afforded complete control of the infection. After four weeks, however, the drug was withdrawn. Thereafter, in the treated animals, lesions developed indistinguishable from those that had been observed previously in the control group. The drug, therefore, did not sterilize the inoculated cornea, but produced an effect similar to that previously observed with thiosemicarbazone (*Brit. J. Pharmacol.*, 1951, **6**, 83).

Clinical Trial.—We have compared two similar groups of patients suffering from long-standing fibro-caseous disease. One group received I.N.H., and the other exactly similar capsules containing lactose. During a six-weeks observation period the group on I.N.H. showed striking subjective and objective clinical improvement, with decreasing cough and sputum, control of pyrexia, increase in appetite, and significant gain in weight. The E.S.R., however, while falling slightly in the treated group, did not parallel the clinical improvement.

The *in vitro* observations on the development of resistance, and the breakdown occurring in experimental tuberculosis on cessation of treatment in some animals, stress the need for caution. We do not therefore propose, despite the encouraging picture shown at the present moment, to publish the full results of the clinical trial until the patients have been under observation for a longer period.

It is quite possible that I.N.H. may have an important place in the chemotherapy of tuberculosis because of its specificity and its initial effectiveness in very low concentrations *in vitro*. On the other hand, it looks as though the problem of resistance may soon be at least as serious as with streptomycin if the drug is indiscriminately used. We feel, therefore, that every effort should be made to find effective combinations of it with other drugs or antibiotics in the hope of delaying the emergence of resistant strains.

We are grateful to Herts Pharmaceuticals Ltd. for the *iso*-nicotinic acid hydrazide used in these investigations.

—We are, etc.,

Departments of Bacteriology, Medicine, and Pharmacology, Guy's Hospital, London, S.E.1.

R. KNOX.
K. S. MACLEAN.
J. M. ROBSON.

Medical Treatment Abroad

SIR,—The increased shortage of foreign currency owing to the prevailing economic difficulties has compelled the Treasury to ask its Exchange Control Medical Advisory Committee to reconsider the policy under which currency certificates are granted to patients seeking treatment abroad.

Until recently, currency was made available for treatment in countries of the European Payments Union in all cases of real illness, whether or not comparable treatment could be obtained at home or within the sterling area. It has now, however, become necessary to adopt a stricter policy, and in future the normal rule will be to allow currency certificates only where the required treatment cannot be had in this country or elsewhere in the sterling area. The Committee will pay regard to the lengths of journey in determining whether or not the sterling area offers practicable facilities. Patients suffering from tuberculosis will usually be allowed currency for treatment abroad, and, during the winter months only, short visits for convalescence after serious illness may be permitted, especially in patients over 65. The rule that a husband or wife is allowed currency to accompany a sick partner abroad, when either of them is over 60, will still apply, and currency may also be allowed for companions in other circumstances, where the Committee considers that it is essential for them to go with the patient.

Under the new procedure the number of applications approved by the Committee is likely to be smaller, but it is satisfied that under the revised arrangements it will still be possible for any person who is seriously ill to obtain any foreign treatment which is essential to recovery.

The Committee's procedure is simple and elastic. The patient's doctor sends the application, with particulars of the case, direct to the Secretary, Exchange Control Medical Advisory Committee, Tavistock House North, Tavistock Square, London, W.C.1, and each application is considered individually upon its own merits. The Committee, which is composed of medical consultants, bases its recommendations, within the above limits, solely upon medical grounds.

The members of the Committee continue to be grateful to the medical profession for their support in this difficult task. Doctors are most helpful in presenting only suitable cases, and when, unfortunately, applications have to be declined the Committee fully realizes that the hard task of explaining the situation to the patient generally falls upon the doctor.—I am, etc.,

HARLEY WILLIAMS,
Secretary.

Admissions to Mental Hospitals

SIR,—I am grateful to Dr. W. Gordon Masefield (April 26, p. 921) for pointing out the error in my second article on "The Psychiatric Case and the Law." Instead of "over half the patients in mental hospitals are voluntary patients," I should have said "over half the admissions to mental hospitals."

I most heartily agree with his view that some simplification of the law relating to the admission of patients to mental hospitals will be necessary as soon as it is seen what effect in practice the National Health Service Act, Ninth Schedule, has had on the procedure in use before it was passed. The case for some form of temporary commitment until the course of the illness is no longer in doubt is a strong one. It is also unfortunate that it is still often necessary to certify patients over 65 years of age.

I feel, however, that no one is more sensitive to present needs than those, like Dr. Masefield, who are concerned with, directly or indirectly, putting the facts to the legislators. The dangers of any hastily prepared legislation on a subject about which so much public prejudice exists are also very much to be borne in mind.—I am, etc.,

Newcastle-upon-Tyne. ALEXANDER KENNEDY.

Discharge of Certified Mental Patients

SIR,—Dr. C. R. Birnie (May 3, p. 971) expresses dissatisfaction over the facility which relatives now enjoy for discharging certified patients from mental hospitals even contrary to medical advice, and he suggests that this facility, which was introduced in 1948, "makes nonsense of the complicated machinery of certification."

I think that Dr. Birnie makes too rigid an interpretation of the law in barring the discharge only of those patients with violent propensities. I maintain that the terms "dangerous and unfit to be at large" permit of a wider interpretation, and indeed with little exercise of ingenuity one could apply those terms to any certified patient who had not fully recovered. Anyway, the final decision rests with the management committee, and the medical superintendent's responsibility in the matter is of limited duration unless he has been so unwise as to let himself be elected to membership of the committee.

Alteration to the existing lunacy laws, as suggested by Dr. Birnie, will not alter the fact that a certain number of mental patients have to be detained against their will. In my opinion the public associate the stigma of mental disorder more with the detention factor than with the business of certification, and that has certainly appeared to be the case since relatives were given facility to discharge patients without all the formalities, delays, and other ordeals necessitated by the former system of discharge by the visiting committee.

My own hospital colleagues and I are conscious of the clinical prestige we have gained by having machinery by which we can advise and effect the immediate discharge of certified patients with the co-operation of their relatives. The same factor has led to a more ready exchange of views between us and patients' visitors, and maybe for that reason I have never had occasion to use stronger methods than advice in dissuading someone from removing a patient unfit for discharge.—I am, etc.,

Shenley, near St. Albans, Herts. OTHO FITZGERALD.

Subconvulsive Dosage in Shock Therapy

SIR,—I should like to draw attention to a statement in the annotation on "Acetylcholine Treatment in Psychiatry" (May 3, p. 964). The author points out that the "efficacy of such treatment is surprising in view of the well-confirmed lack of success of electric-shock treatments which fail to cause a major seizure, and it is not clear why acetylcholine should be effective in subconvulsive doses." This statement may well be misleading and give the impression that the actual physical convulsion is a necessary part of the mode of action of the treatment. Subconvulsive treatment is generally very unpleasant to the patient and causes considerable confusion afterwards, especially in the elderly and arteriosclerotic. It is therefore rarely persisted in by those administering the treatment, and it is found in practice that if a "missed" fit does occur the expected confusion can be minimized by immediately increasing the treatment dose to produce a major convulsion. It should not, however, be thought that the actual clonic movements so induced play any part in the efficacy of the treatment, which would seem to be solely concerned with the intensity with which the cortex is stimulated.

Whether subliminal stimulation would be equally effective is surely rarely put to the test, because in practice it is rarely persisted in. It is certainly not to be advised in electric-shock therapy, anyway, for the reasons I have given. It would, however, be regrettable if the impression were given to the unthinking that a lot of jerking was necessary before E.C.T. could be effective; indeed, this has been fully disproved, as the treatment is equally effective when all movement has been abolished by the use of curare or similar agents.—I am, etc.,

Oxford. HUGH F. JARVIE.

Sister Kenny

SIR,—I was interested in reading Mr. H. J. Seddon's review of Sister Kenny's book *And They Shall Walk* (April 12, p. 802). Whilst he admits that her influence on the treatment of poliomyelitis "has been thoroughly healthy" and points out that the book was intended chiefly for laymen, he goes on to discuss some technical points.

It is encouraging that he admits the accuracy of her observation of what she terms "spasm," for, although this appears to be fairly universally accepted in the United States, there still appear to be many in this country who fail to appreciate it. But it appears that even Mr. Seddon fails to understand its full significance, for, although he goes on: "If what is sound in her practice was universally practised today we should be well on the way to abolishing one of the most serious consequences of paralytic disorders—namely, deformity," yet hundreds of patients are still seen in this country for whom little appears to have been done to relieve this condition referred to as "spasm." Yet it or its sequence is still present if one appreciates how to detect it, and even after 18 months or more can be overcome, though much more laboriously, if adequate means are used.

Having had the privilege of touring the United States and spending some months visiting hospitals in various parts of that country—including three of the Elizabeth Kenny Treatment Centres in Jersey City, Minneapolis, and California—I had the good fortune of not only meeting Sister Kenny at her work (when I learnt much about poliomyelitis from her), but also of meeting her in her home and in the homes of others.

That there may be some people in America who were glad to see Sister Kenny retire may be true, but her retirement from that country occurred whilst I was still there, and, if what I saw was any indication, quite the opposite appeared to be the case. And it may also interest many to know that she has recently been asked to return to the U.S.A. to further investigations on changes in the skin.—I am, etc.,

London, W.1. LANCELOT H. F. WALTON.

Local Aureomycin for Aphthous Stomatitis

SIR,—Following a report (Distelheim, I. H., and Sulzberger, M. B., *J. invest. Derm.*, 1949, **13**, 115), we have tried the effect of local "aureomycin" in the form of an aqueous solution, with encouraging results, in aphthous ulceration of the mouth.

We have treated eight patients. A 250-mg. capsule is dissolved in 2 oz. water and the solution used as a mouthwash four or five times a day after meals. The solution is held in the mouth for about five minutes. A fresh solution is prepared daily. In recurrent cases treatment is started as early as possible. In all cases healing has occurred within seven days of starting therapy, and some within a much shorter period. In one—a severe recurrent case—the whole of the inside of the lower lip was deeply ulcerated. In the recurrent cases subsequent attacks have responded rapidly to the same treatment.

Other successes have since been reported from America. Fisher and Leider (*Arch. Derm. Syph., Chicago,* 1950, **64**, 164) treated eight cases with aureomycin troches each containing 15 mg. of the antibiotic. Six of the eight benefited, but two got "aureomycin mouth." Everett (*J. Amer. dent. Ass.*, 1950, **40**, 555) treated 38 patients with benefit in all but five cases. The average duration of the lesions fell to 6½ days as compared with 10 days in a control series. Systemic therapy has also been reported as successful in one case (*J. invest. Derm.*, 1949, **13**, 51).—I am, etc.,

Department of Dermatology, United Bristol Hospitals. EDWARD PRETTEJOHN.

Xylocaine

SIR,—For some time we have followed with interest the comments on Dr. P. R. Bromage's letter (February 9, p. 328) concerning a fatality following the use of "xylocaine." We have administered this drug in over 1,000 varied procedures ranging from topical and local infiltration to continuous peridural analgesia for thoracic and abdominal surgery. Beyond doubt, this drug is superior to any other local analgesic drug in clinical ability and overall effectiveness.

It is our belief that the concentrations of xylocaine solutions used for regional analgesia are unnecessarily strong. For the past 50 years procaine has served as the universal local analgesic drug, and the volumes and concentrations used in all techniques of regional analgesia have been adapted to the activity and toxicity of this drug. It is reasonable to assume that, if a drug of greater toxicity is substituted for procaine in similar volumes and concentrations without regard to toxicity and activity of the substituted drug, a greater incidence of reaction will occur.

The following facts must be considered before a dosage of xylocaine is selected. (1) In concentrations of 0.5% or less the toxicity of xylocaine is approximately that of procaine. However, in solutions of 1% the toxicity of xylocaine is 40% greater, and in 2% solutions it is 50% greater. This indicates that in dilute solutions the problem of toxicity is considerably less than with stronger concentrations. (2) The activity of xylocaine is three to four times that of procaine, and therefore should be used in lower concentrations. (3) The exhibition of xylocaine results in a greater area of analgesia than with a similar volume of any other local analgesic drug. It may therefore be concluded that xylocaine should be used in far lower concentrations and volumes of solution than procaine and related compounds. We have found that a solution of 30 ml. 0.8% xylocaine with adrenaline is best for peridural analgesia in thoracic surgery. However, we have used 0.5% solution with satisfactory results in an effort to find a minimal effective strength of this drug for this technique. We also believe that 0.25% is adequate for infiltration analgesia and that the volume should be approximately two-thirds that of procaine used for a similar technique. The total volume should never exceed 100 ml. within one hour or 200 ml. for a surgical procedure. Adrenaline should be added to large volumes of solutions to prevent rapid absorption, provided its use is not contraindicated, and it rarely is. A 1% solution is adequate for the majority of regional blocks.

—We are, etc.,

Springfield, Missouri. ORAL B. CRAWFORD.
ALLEN BAILEY.

General Anaesthesia in Ophthalmic Operations

SIR,—In his letter (April 5, p. 762) Mr. John Foster refers to the question of general anaesthetics for eye operations, and particularly mentions cataract operations. At Southport Infirmary I have been doing practically all major ophthalmic surgery under general anaesthesia since April 14, 1951, with most gratifying results. Children are anaesthetized first with rectal "avertin," and anaesthesia is thereafter maintained with gas, oxygen, and "trilene"; adults are anaesthetized with thiopentone, or thiopentone and curare.

The majority of the anaesthetics have been given by Dr. Raymond Smith, and we are preparing a paper for publication on this subject.—I am, etc.,

Southport. DAVID RANKINE.

Explosion During Anaesthetic

SIR,—Dr. J. F. Burdon's suggestion (May 3, p. 972) that inflammable gas arising in the stomach might be the cause of an unexplained anaesthetic explosion recalls a memory of my student days.

One medical ward round at King's College Hospital was much enlivened when the discussion on a case began with the history that the man had been at a theatre, and in the act of lighting a cigarette had suddenly exploded. After an appropriate interval for recovery, the class proceeded and reached a diagnosis of pyloric obstruction with gross gastric retention. It was presumed that fermentation of the stomach contents had formed inflammable gas, and that the approach of the light to the cigarette had coincided with a most inopportune belch. So far as I can remember the man was admitted to King's under the care of Dr. Terence East in 1929 or 1930, and it may well be that the case has been reported already.—I am, etc.,

Maryport Cumberland. J. D. H. BIRD.

Congestive and Simple Glaucoma

SIR,—I most emphatically endorse Mr. H. E. Hobbs's opinion, expressed in his letter (April 19, p. 869), on the predominant importance of perimetry and scotometry in the early diagnosis of lesions of the visual pathways. So far as the changes in the optic nerve peculiar to glaucoma are concerned, however, my conception of their pathology is such as to lead me to believe that these disturbances are inextricably associated with anatomical changes in the optic disk, which, even in the earliest stages of the disease, are ophthalmoscopically evident. Unless incontrovertible evidence to the contrary should prove me wrong, I shall not fear failure to recognize cases of early glaucoma simplex by my omitting to include scotometry in my routine examination of middle-aged and elderly persons the appearance of whose disks is above suspicion.—I am, etc.,

London, W.1. R. AFFLECK GREEVES.

Epidemiology of Hypertension

SIR,—Your leading article (May 3, p. 962) comes at an opportune moment, for it is becoming apparent that a high diastolic blood pressure is a relatively common physiological deviant in the inhabitants of western societies, whereas on the aetiological side, to quote Professor John McMichael (p. 933), "stupendous efforts . . . have produced an ever-growing halo of light which merely illumines an ever-increasing sphere of darkness." This is a frank but very much needed statement of our fundamental ignorance about causation and contributory factors. It opens our minds to the wider possibilities contained in the concept that hypertension is a stress disorder—i.e., a pattern of psychophysiological strain in the organism, as a response to environmental pressure. A study of the psychosocial situation of the hypertensive is obviously indicated. For those who propose to explore this promising field there are already several pointers to be got from the literature devoted to psychosomatic medicine. I have recently reviewed this elsewhere (*Brit. J. med. Psychol.*, 1951, **24**, 242), and propose to draw attention to certain pertinent findings.

(1) There is a well-defined character structure, about which all investigators are in agreement. For the moment, this character structure can be taken to apply only to those patients who develop the many symptoms of anxiety type that cannot be related to the organic consequences of arterial hypertension. It reveals the remarkable subjective difficulties that bedevil the hypertensive's relationship with his fellows and with the members of his family.

(2) There are typical psychosocial situations in which the hypertensive falls ill with symptoms (including encephalopathy). Several investigators have shown how rewarding can be a parallel exploration of symptom onset and of biographical events of

intense emotional significance. He develops symptoms in relation to his employer, his wife, or his mother, and not, as is so often asserted, primarily as an anxiety response to being told that his blood pressure is high.

(3) There is evidence of a direct correlation between the level of diastolic blood pressure and a current emotional situation (Hambling, J., *Brit. J. med. Psychol.*, 1952, **25**, 39). Under certain conditions of interview it is possible to map out the "pressor areas" in the patient's emotional life by the fluctuations that occur in his diastolic blood pressure during his account of himself. This can happen with patients in each of the three phases of hypertension—i.e., "diastolic reaction," benign hypertension, malignant hypertension—and applies to asymptomatic as much as to symptomful cases.

(4) There is suggestive evidence, as yet unpublished, that lack of affection, severity of treatment, and violence between parents may be the childhood background to the character difficulties described in the literature.

Questions of ultimate aetiology apart, it will be right to explore fully the characterology and psychosocial situation of the hypertensive, because it promises to lead to advances in management directed towards minimizing the pressor effect of the environment. It may be eventually found that young diastolic reactors can be "desensitized" so that they no longer react. Eventually, if the childhood situation turns out to be a significant factor, then the therapy of hypertension enters the field of general preventive medicine in the sector of the mental hygiene of the family group.—I am, etc.,

London, W.1. JOHN HAMBLING.

Congenital Absence of Rectum

SIR,—While agreeing with most of Dr. Isabella Forshall's remarks (April 5, p. 762), I cannot agree that gas will be seen in the terminal hind gut on radiography within six hours of birth. In most cases almost 24 hours must elapse before gas passes through the sticky green-black meconium in the terminal gut. If the radiographic appearances at six hours are taken as indicating accurately the termination of the hind gut, not only will the abdomino-perineal approach be used needlessly in cases in which only a thin diaphragm separates the hind gut from the surface, but the operator will find himself in considerable difficulty in attempting to follow the hind gut below the peritoneum in the diminutive pelvis of the infant. Admittedly a fistulous connexion with the urinary tract may be found and divided.

As Miss Forshall suggests, the combined abdomino-perineal approach for high rectal atresia is practised in other centres—for the past three years in the Royal Hospital for Sick Children, Glasgow. The mortality in this apparently "major" procedure performed during the first few days of life is considerably lower than the mortality following a "simple" colostomy performed in the newborn. With the exception of the case with recto-vaginal fistula, colostomy should rarely be required in this condition. A high mortality in this condition is related either to delay in sending patients to a suitable centre of paediatric surgery or to other associated severe congenital defects.—I am, etc.,

Glasgow. W. M. DENNISON.

Is Latin Dead?

SIR,—The following story is taken from *The Last Serjeant*, by Serjeant A. M. Sullivan, Q.C. There was a judge much given to Latinity who once asked counsel: "Has your client never heard, *Sic utere tuo ut alienum non laedas*?" To which counsel answered: "Not a day passes, your Honour, on which he does not hear it. It is the sole topic of conversation where he lives at the top of Mushera Mountain."—I am, etc.,

Bristol. WALTER SIMPSON.

SIR,—Dr. J. Lawrence Henry (April 26, p. 924) pleads for a continued use of Latin in medicine, but, while the study of Latin at school is a stimulating mental process, the reading, writing, and, most of all, the speaking of it demand great erudition. A medical student cannot reasonably be expected to do justice to the majestic Latin tongue any more than an itinerant musician could be expected to play the Caprices of Paganini on a tin whistle.

Actually, English is taking the place of Latin as the international language. In Europe and the Scandinavian countries English is a compulsory school subject, and it is taught in such a way that pupils are eventually able to speak it intelligibly and understand it when it is spoken. In this country the acquisition of a working knowledge of foreign languages is grievously hindered by the waste of valuable time on the study of the minutest details of grammar and syntax, and after years of drudgery at school little practical progress is made.

I make bold to say that foreign doctors would prefer good English to bad Latin. Let us use English, therefore, lest we make Cicero and Virgil stir uneasily in their graves.—I am, etc.,

Strichen, Aberdeenshire. WILLIAM HARKINS.

SIR,—May I as a British bilingual practitioner with British and Continental qualifications support the plea of Dr. J. Lawrence Henry (April 26, p. 924) for the revival of the Latin language among medical men? While in most other languages, in particular German, practically all anatomical and medical terms have indigenous equivalents which are commonly used among the lay population, and only medical men use an unadulterated Latin or Greek nomenclature in their professional language, the English language is unique in that it lacks Anglo-Saxon terms for many of these, and anglicized Latin or Greek expressions, always pronounced according to English phonetics, are used equally by laymen and professionals. Larynx, duodenum, diabetes, pleurisy, are only four examples of a big list of terms without accepted native equivalents, while gullet, the English name for oesophagus, is unfamiliar, and the midriff means to the modern layman not the diaphragm but the unclothed portion of the dancer's or artiste's body between blouse and skirt. A limited knowledge at least of Latin and Greek (most medical terms are derived from the latter rather), by no means a scholastic delving into the writings of Hippocrates and Galen, will always stimulate the scientific interest and insight of the medical man lest he forget that medicine is not only the most realistic, but also the most humanistic of all sciences. He will then do justice to the popular appraisal of his art and learning, which accords him alone among the professions the noble title "doctor"—i.e., wise man—without implying an academic distinction.

I much regret, however, Dr. Lawrence Henry's disparaging remarks about the metric system. Surely this is not advocated to help foreign readers, but to bring medicine and pharmacy in line with all the other sciences, which have long ago abandoned the foot-pound-duodecimal for the centimetre-gramme-decimal system. The medical profession here lags sadly behind, and might be accused of playing down to popular tradition at the expense of scientific accuracy. The grain and minim should be entirely abandoned in favour of the gramme and cubic centimetre.—I am, etc.,

Leicester. A. K. GRAF.

POINTS FROM LETTERS

Debauchery of Honest Words

Dr. WILLIAM FOSBERY (Batheaston) writes: I trust that Dr. C. W. M. Whitty (April 5, p. 762) would not advocate the use of such neologisms and new-coined phrases as the following, which I saw recently in the U.S.A.: "The basement is closed tonite, as it is being groceteriarised."

Correction

Mr. C. LANGTON HEWER writes to point out that in his letter on succinylcholine (May 3, p. 971) the dose of succinylcholine recommended by the makers for a large adult should have read 100 mg.

Obituary

MARIA MONTESSORI, M.D.

Maria Montessori, who died on May 6 at the age of 81, was an Italian pioneer in the education of young children. Her principles of teaching, which were revolutionary, like those of Pestalozzi and Froebel, with whom she may be compared, gained acceptance in many countries.

Dr. Montessori came into pedagogy by way of medicine, in which she graduated in 1904, being the first woman to receive the M.D. degree of the University of Rome. She became an instructor in a psychiatric clinic in that city, and on applying herself, about 1907, to the treatment of mentally defective children she came to the conclusion that in such cases the teaching approach was more important than the medical. Her system of free discipline and sensory training proved so successful that children of very low mentality were enabled to pass the State examination in reading and writing ordained for normal children. Like Freud, she then applied the experience gained in abnormal cases to normal children, arguing that, if the mentally deficient could by educational means be brought up to the level of normal children, still more startling results might be gained with children normal from the beginning. She adapted her methods to older children, though here she was less successful, perhaps because of more resistance from orthodox teachers. At all events, children from 3 to 6 seemed to profit more from the Montessori method than children from 6 to 10.

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The two important Montessori principles were non-interference with the child's freedom and individuality and the use of sensory training in the earliest stages of education. Dr. Montessori's contention was that the best kind of education was learning for oneself, with the teacher as an observer, and that any necessary discipline arose naturally from the teacher's personality. Individual separate activity for each child was a distinctive feature of her system, in contrast to the traditional custom of teaching large classes in unison, and every child had freedom to work at his own pace and to select his own material. Success in these endeavours led to Dr. Montessori's appointment as lecturer in pedagogic anthropology in the University of Rome and to the establishment of all-day schools for young children in the slum quarters of the city. She published several works elaborating her method, and she travelled widely, giving lectures and demonstrations. Societies were formed, in England and other countries, to explore and make known the Montessori technique, training colleges and research institutes were established, and annual congresses held. There are 200 Montessori schools in this country, and only last year Dr. Montessori attended a congress in London and impressed all who saw her by her handsome appearance in old age and her vigorous personality. She had already received academic recognition in this country, the honorary degree of Doctor of Literature having been conferred on her by the University of Durham in 1923.

During Mussolini's regime in Italy Dr. Montessori was at first patronized by the Italian dictator; but she fully appreciated the dangers of such patronage and left Italy in 1932. Her schools were banned thereafter. The Montessori movement, owing to its racial and religious tolerance, also came into disfavour in Hitlerite Germany. When the second world war broke out Dr. Montessori, who happened to be in India at the time, was interned for some months as an enemy alien.

Her work as an educational reformer must not be judged by the number of schools to which the name Montessori is attached; her influence in education and child psychology has had a much wider reach, and the positive aspects of her work have been adopted in many fields where her name is not acknowledged.

J. J. LEVIN, M.B., Ch.B.

The death early this year of Dr. J. J. Levin has removed an outstanding personality from the South African medical world. Joseph John Levin was born in Cape Colony on February 19, 1885, the son of the late Mr. Joseph Levin, and was educated at the Marist Brothers' School, Johannesburg, and at Victoria College (now the University of Stellenbosch) until 1905, when he entered the University of Edinburgh as a medical student. He graduated M.B., Ch.B. in 1910, a year notable for its South African graduates of the university. He then held house appointments at the Royal Infirmary, Edinburgh, and at the Royal Southern Hospital and the Maternity Hospital, Liverpool, and was obstetric assistant in the University of Liverpool. During the war of 1914–18 Levin served as a captain in the South African Medical Corps in the campaign in German South-West Africa and was mentioned in dispatches. After the war he was in practice in Johannesburg, where he became well known as a surgeon and as a medico-legal expert. He was appointed honorary assistant surgeon (later honorary consulting surgeon) to the General Hospital and junior lecturer in clinical surgery and lecturer in charge of the department of forensic medicine in the University of the Witwatersrand. Always a very loyal and active member of the South African Medical Association and of the British Medical Association, he played an important part in the negotiations which led to the old South African Medical Association becoming affiliated to the B.M.A. He was honorary secretary of the Witwatersrand Branch from 1919 to 1921, chairman of the Johannesburg Division, and president of the Southern Transvaal Branch in 1929–30. He was a member of the Federal Council almost from its inception in 1928 and served on it continuously until 1946, when ill-health compelled him to resign. He was also a member of the executive committee from 1941 to 1946. For his long and faithful service he was awarded the bronze medal of the South African Medical Association in 1950.

The following is an extract from the appreciation of Dr. Levin written by Dr. A. W. S. Sichel, President of the British Medical Association, and published in the *South African Medical Journal* of March 15: The passing of Joseph Levin removes from the stage of the Medical Association one who played his part in vigorous fashion over a long period. He will be remembered more particularly for the yeoman service he

rendered on the Workmen's Compensation Committee, of which he was convener for several years. He acted as organizing secretary of the medical congress held at Johannesburg in 1931, on which occasion, with the help of his megaphone, he dominated the proceedings most effectively, as many will recall. Again in 1938 he displayed remarkable activity as organizing secretary of the congress held at Lourenço Marques. My first acquaintance with him covered the years 1907 to 1910, when we were fellow-students at the University of Edinburgh and played together in the university first fifteen. In rugby, as in later years in medical practice, his methods were robust rather than polished, but he always gave of his best, though in his own individual manner. Our association on the Federal Council in later years caused our earlier acquaintance to ripen into a friendship which became all the closer when ill-health laid him low. The memory of his rugged personality will not be forgotten by his contemporaries, nor will his work for the Medical Association go unappreciated by those with whom he worked in the conduct of its affairs.

G. B. MOWER WHITE, F.R.C.S.

Mr. Gilbert Benjamin Mower White, who died at his home in Buckinghamshire on April 27 in his 87th year, was born on March 29, 1866. His family lived at Romsey, Hampshire, and he was educated at Monkton Combe School and at University College Hospital, qualifying M.R.C.S., L.R.C.P. in 1888. He took the London M.B. in the same year and the B.S. in 1889, both with honours. He became F.R.C.S. in 1891. After holding the usual resident posts he was appointed demonstrator of anatomy at University College Hospital, a position he retained for many years. In 1893 he was elected surgeon to the Great Northern Central Hospital, which became the hub of his surgical life. At that time the hospital was rather the temporary home of migrants—W. H. Cripps, G. H. Makins, Raymond Johnson, Percy Dean, C. B. Lockwood, Herbert Allingham, Warren Low—who all stayed awhile and then departed to other fields. Not so Benjamin Mower White. He found in the Royal Northern Hospital, as it became known later, an opportunity of congenial surgical work that suited his temperament. In the course of time he developed a large surgical practice in north London and gathered about him a devoted band of doctors and patients. His additional appointments to the Mildmay Mission and Willesden General Hospital left him little time for original work, and few publications came from his pen. During the war of 1914–18 the depletion of the surgical staff threw a great burden on him personally. For his care of the many wounded soldiers who passed through his hands he received a special mention. As an operating surgeon he was methodical, unruffled, and sound in technique; slow, perhaps, but the slowness was not the result of indecision—only an indication of the extreme care with which he worked. He could operate speedily if need arose. In committee his wise judgment was often sought, and for some years he was chairman of the house committee of the Royal Northern Hospital, a post usually reserved for a layman. His personality was a delightful one, for the twinkling eye and the cherubic countenance were no mask. Affectionately known as Benjamin, he was beloved by all. In 1920, when he was due to leave the staff of the hospital, his lay and medical colleagues created the post of emeritus surgeon, which he held, with the care of beds, for another five years, when he was appointed consulting surgeon. For some years after he had left the hospital he continued active surgical practice. He leaves a widow, two sons, and four daughters, one of whom is in the medical profession. —L. E. B.-W.

Dr. FREDERICK JOHN CUTLER, who died on April 28, received his medical training at Guy's Hospital, qualifying M.R.C.S., L.R.C.P. in 1909. He had practised in Hastings for over 30 years, settling in the town at the end of the war of 1914–18, in which he served as a regimental medical officer in the R.A.M.C. He obtained a number of medical appointments and was honorary surgeon to the Buchanan Hospital, St. Leonards-on-Sea, for some years. He was an active member of the British Medical Association, being chairman and later secretary of the local Division, secretary of the local medical and panel committee, and its representative at the Annual Conference. Dr. Cutler was an able clinician, well endowed with common sense, never given to panic, averse to hasty decisions, and very popular with the patients in the large practice which he built up in the years between the two world wars, a practice in which he was joined by his son, Dr. M. J. Cutler. He had a strong sense of humour, and his cheerful kindliness gave his patients great confidence in times of crisis. Only his more intimate friends knew how much anxiety a critical case would give him, or how much his peace of mind was disturbed by his patients' troubles. To his wide circle of friends "Freddie" Cutler was well known as the best type of sportsman. No ball game came amiss to him. He played rugby and tennis for Guy's, and was well known in Hastings on the tennis court and golf course. He was a generous winner, a good loser, and a most formidable opponent if the game was going against him. Of late years his health had begun to fail, and he found it increasingly difficult to stand the rigours of the English winter. He finally took to going to sea in the Merchant Service in the autumn until the following spring, and found that the warmth and sunshine of the Tropics gave him complete relief and enabled him to carry on some medical work under ideal conditions for his state of health. It was hoped that this improvement would become permanent, but he died at sea after a short illness.—W. G.

Medical Notes in Parliament

HEALTH BILL IN THE LORDS

In the House of Lords on May 6 the National Health Service Bill came up for Second Reading. Lord WOOLTON explained its proposals.

Lord WEBB-JOHNSON said he would go even further than the Government. He could not understand why in-patients should be exempt from charges. On the average they were better able to pay than other people who made claims on the National Health Service.

Lord HAWKE said a health service was bound, if it was entirely free, to cost more and more. If all sufferers from rheumatoid arthritis were treated free with "cortisone" the Bill might go up by millions of pounds per year.

The Bill was read a second time without a division.

Independent Inquiry at Broadmoor

Mr. H. F. C. CROOKSHANK, on May 6, announced that the Government had decided that the recent incident at Broadmoor raised problems which required further independent

inquiry. Mr. Crookshank said he had inspected Broadmoor six weeks ago, and also much earlier, when he had been Under-Secretary to the Home Department. He had not found that transfer from the Home Office to the Board of Control under Section 62 of the Criminal Justice Act (1948) had resulted in any diminution of the precautions previously taken.

Mr. IAIN MACLEOD announced on May 12 that the following people had agreed to undertake the inquiry: Mr. J. Scott Henderson, Q.C. (Chairman); Captain C. Waterhouse, M.P.; Mr. Kenneth Younger, M.P.; Dr. P. K. McCowan.

The Minister promised to keep the House informed as fully as possible of their findings, but pointed out that the whole of the committee's report could not be published, as security arrangements were involved.

Cars by Hire-purchase for Doctors

On May 8 Mr. C. W. BLACK asked whether the President of the Board of Trade had considered the application made to him on behalf of doctors for some relaxation in the restrictions on the hire-purchase of motor-cars.

Mr. PETER THORNEYCROFT answered that to assist doctors in their service to the public he was willing to consider applications for licences from doctors in or entering general practice who did not own and had not recently owned a car, allowing them to pay off the balance of the purchase price in a longer period than the maximum of 18 months laid down in the Hire-Purchase and Credit Sale Agreements (Control) Order, 1952. Applications would not normally be considered in respect of cars costing more than £800. The maximum period for repayment would be three years. Doctors who satisfied these conditions could apply for licences through their professional organizations or direct to the Board of Trade.

Foot-and-mouth Disease

Answering, on May 8, numerous questions about foot-and-mouth disease, Sir THOMAS DUGDALE said the situation was serious, but it was not unprecedented. Unless it deteriorated seriously, it could be brought under control. Since November, 1951, until May 8, 1952, there had been 258 outbreaks of the disease in this country. This should be compared with the experience of Denmark, where 26,000 outbreaks occurred in the last six months, and that of Holland, where there had been 23,000 in roughly the same period. In Western Germany the number of outbreaks in 1951 was 155,000. The outbreaks that occurred in the United Kingdom in the winter of 1937–8 and in 1941 were comparable in severity to the present one. In every case the disease was stamped out. Research on foot-and-mouth disease was carried out at the Foot-and-Mouth Disease Research Institute at Pirbright. £93,540 had been provided for grants to this institute for the current financial year, and increased funds were being made available for the development of the station. The governing body would in future be publishing an annual report. The virus of the present outbreak had been identified as type "A."

Cruelty to Children

Squadron Leader A. E. COOPER raised, on May 7, the subject of cruelty to children.

Mr. SOMERVILLE HASTINGS said that in every area there should be some authority well known to the people to whom they could report cases.

Sir HUGH LUCAS-TOOTH said that from 1948 to 1951 the N.S.P.C.C. investigated about 40,000 cases of alleged cruelty each year. In 1951 altogether 1,076 persons were found guilty under Section 1 of the Children and Young Persons Act, of whom 30 were convicted on indictment. This figure had been roughly stable over a number of years. Ignorance was easily the largest cause of cruelty, the people concerned being definitely of subnormal intelligence. The most practical type of remedy was to train mothers found guilty of neglect. Classes in home management were provided in Holloway and Manchester prisons. There were also homes where, on a medical recommendation, mothers might be sent, accompanied by their children up to the age of 7. The Home Office thought that existing powers of punishment were adequate and were adequately applied by the Courts. About 75% of local authorities had taken steps to implement the joint circular of July 31, 1950. In the event of suspected cruelty to a child, the proper person to tell was the nearest policeman.

Allowances and Pensions

Mr. OSBERT PEAKE, on May 5, moved the Second Reading of the Family Allowances and National Insurance Bill. He said its aim was to restore to National Insurance the principle of uniformity in benefits between sickness, unemployment, and widows' pensions, and retirement pensions at minimum retirement age.

Mr. I. N. MACLEOD said that in 25 years' time the scheme would be losing £470m. a year. That was more than was spent now on the whole of the National Health Service Scheme. It was almost entirely the burden of retired pensions that the House had to study, and this Bill was adding £36m. to them.

Mr. R. H. TURTON, replying for the Government, promised that consideration would be given in due course to the proposal to make improvements in the system of maternity allowances.

The Bill was read a second time, without a division.

Lead Poisoning

On May 8 Miss ELAINE BURTON raised the subject of lead poisoning in children. There was a danger of lead poisoning in the application of powder, containing lead, to the skin, but she had been unable to find out how much of the powder sold for infant skins contained lead. A second source of danger was the use of lead nipple shields by mothers. Details of five cases had been published in the *British Medical Journal* of February 17, 1951 (p. 329). She asked whether the Board of Trade would consider recommending that paint containing lead should never be used in places that children could reach, and that lead nipple shields should be prohibited.

Mr. HENRY STRAUSS, for the Board of Trade, said the children under 5 in this country whose deaths could be traced to lead poisoning were very few. His department and the Ministry of Health could trace only six cases in the last 20 years. The view of Dr. Sheldon, consultant adviser in paediatrics to the Ministry of Health, was that this was rare and becoming rarer. He came across only about two cases a year. In no case had it been established that the original manufacturer was to blame. The larger manufacturers of toys certainly did not at present use lead paint. It was true that baby powders were dangerous in some countries, but on inquiry he had not found they were so in this country. Lead nipple shields were undoubtedly dangerous. The Ministry of Health had warned hospitals in 1949 of the dangers of issuing them to nursing mothers. It had been arranged that family doctors and chemists should be warned. These shields were excluded from appliances which doctors might prescribe under the National Health Service. Distributors stopped the sale of such shields, and a warning notice was sent to the general press. These actions had been completely effective. He did not think that a case had been made for further legislation.

General-practitioner Hospitals

Mr. IAIN MACLEOD stated, on May 12, that there were 386 general-practitioner hospitals (excluding maternity homes) on July 5, 1948; 351 of these still existed as general-practitioner hospitals, 21 had become general hospitals, 6 annexes of general hospitals, 5 special hospitals, and 3 general practitioner maternity homes. A number of additional general-practitioner units had been provided elsewhere.

INFECTIOUS DISEASES AND VITAL STATISTICS

Summary for British Isles for week ending **April 26** (No. 17) and corresponding week 1951.

Figures of *cases* are for the countries shown and London administrative county. Figures of *deaths* and *births* are for the 160 great towns in England and Wales (London included), London administrative county, the 17 principal towns in Scotland, the 10 principal towns in Northern Ireland, and the 14 principal towns in Eire.

A blank space denotes disease not notifiable or no return available.

The table is based on information supplied by the Registrars-General of England and Wales, Scotland, N. Ireland, and Eire, the Ministry of Health and Local Government of N. Ireland, and the Department of Health of Eire.

CASES in Countries and London	1952					1951				
	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire	Eng. & Wales	Lond.	Scot.	N. Ire	Eire
Diphtheria	35	4	6	0	2	32	1	12	0	2
Dysentery	275	35	44	1		871	136	104	11	3
Encephalitis, acute	6	0	0	0		6	1	0	0	
Enteric fever:										
Typhoid	0	0	0	1	1	4	0	1	0	0
Paratyphoid	8	0	0	0	2(B)	4	0	0	0	
Food-poisoning	123	5		0		71	5		2	
Infective enteritis or diarrhoea under 2 years				12	16				18	24
Measles*	6,137	275	769	276	446	18,006	1017	482	226	151
Meningococcal infection	46	3	13	2	1	29	2	22	2	6
Ophthalmia neonatorum	49	4	12	0		45	1	8	0	
Pneumonia†	747	47	266	8	2	570	34	197	3	
Poliomyelitis, acute:										
Paralytic	22	3	3	0	1	11	0	2	0	3
Non-paralytic	7	0				8	1			
Puerperal fever§	281	35	16	0	1	85	8	15	2	3
Scarlet fever	796	54	141	12	55	1,084	87	143	32	80
Tuberculosis:										
Respiratory			148	37				188	42	
Non-respiratory			26	1				38	9	
Whooping-cough	2,946	163	235	113	62	3,643	248	555	50	58

DEATHS in Great Towns	1952					1951				
	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire
Diphtheria	1	0		0	0	*0*	0	*0*	0	0
Dysentery	0	0		0			0		0	
Encephalitis, acute		1			0		0			0
Enteric fever	0	0		0		*0*	0	*0*	0	
Infective enteritis or diarrhoea under 2 years	5		4	0	1	*12*		*2*	0	3
Influenza	17	4	5	0	0	*13*	1	*0*	1	0
Measles		0		0	3		0	*1*	0	0
Meningococcal infection		0	1				0	*1*		
Pneumonia	241	26		10	7	*192*	30		15	9
Poliomyelitis, acute	2	0			0	*5*	0			0
Scarlet fever		0		0	0		0	*0*	0	0
Tuberculosis:										
Respiratory	133	19	19	2	10	*127*	15	*20*	6	11
Non-respiratory		1	3	0	1		8	*3*	1	6
Whooping-cough	4	0		0	0	*5*	1	*1*	0	0
Deaths 0–1 year	224	21	45	7	28	*230*	32	*44*	11	22
Deaths (excluding stillbirths)	5,129	744	620	113	202	*5,015*	763	*633*	121	180
LIVE BIRTHS	8,092	1227	973	243	450	*7,190*	1141	*956*	232	457
STILLBIRTHS	204	27	25			*195*	27	*31*		

* Measles not notifiable in Scotland, whence returns are approximate.
† Includes primary and influenzal pneumonia.
§ Includes puerperal pyrexia.

Vital Statistics

Smallpox in Lancashire

The last case of smallpox notified in England and Wales occurred in Middleton Borough, and was removed to hospital on April 17. All districts have now been declared free from smallpox infection. Two convalescent cases remain in hospital and are awaiting discharge.

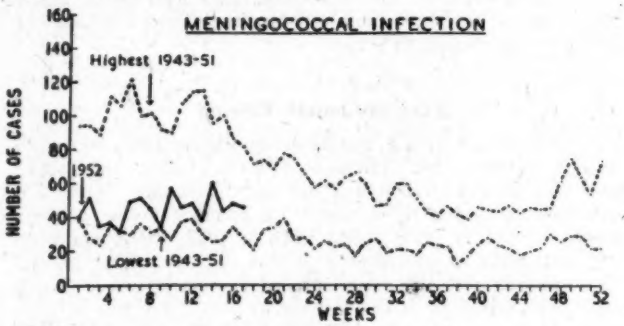
Not Smallpox at Swansea.—A final diagnosis of chickenpox has been made in the patient put ashore at Swansea on April 23 with suspected smallpox (May 3, p. 981).

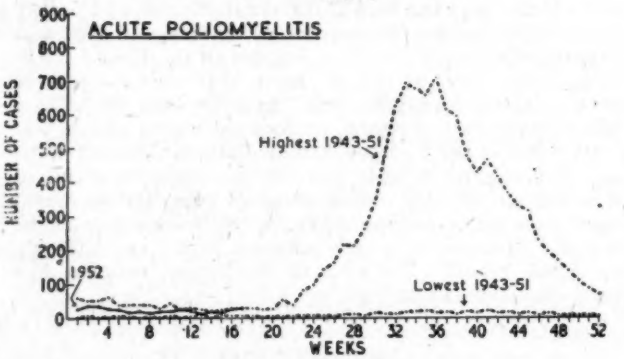
Less Smallpox in Mexico

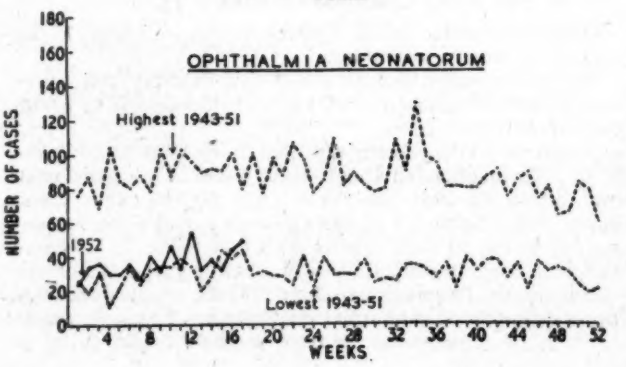
The incidence of smallpox in Mexico has declined notably in the last three years, according to W.H.O. The number of reported cases was 1,060 in 1949, 769 in 1950, and only 25 in 1951. This pronounced decrease is a result of the vaccination campaign which has been carried out by the national health authorities.

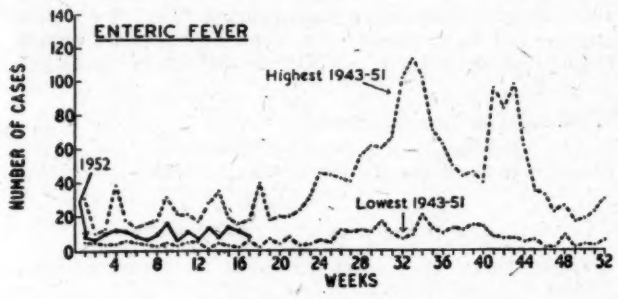
Graphs of Infectious Diseases

The graphs below show the uncorrected numbers of cases of certain diseases notified weekly in England and Wales. Highest and lowest figures reported during the nine years 1943–51 are shown thus - - - - - - - -, the figures for 1952 thus ————. Except for the curves showing notifications in 1952, the graphs were prepared at the Department of Medical Statistics and Epidemiology, London School of Hygiene and Tropical Medicine.









Infectious Diseases

Falls were recorded in the number of notifications of measles by 637, scarlet fever by 247, acute pneumonia by 73, and dysentery by 52 in England and Wales during the week ending April 26, while the only rise of any size was whooping-cough by 146.

The largest declines in the incidence of measles were Lancashire 134, Norfolk 114, Yorkshire West Riding 108; the only large exception to a downward trend was a rise of 136 in Northumberland. The rise in this county was mainly contributed by Newcastle-upon-Tyne C.B. and Wallsend M.B., where the notifications increased by 82 and 42 respectively. A small decline in the incidence of scarlet fever was recorded throughout the country. The largest fall was Lancashire 50. The number of notifications of diphtheria was 3 more than in the preceding week. The chief feature of the local returns of diphtheria was a rise of 6 in Warwickshire. All of the 11 cases in this county were notified in Birmingham C.B. The largest rises in the number of notifications of whooping-cough were London 52, Yorkshire West Riding 52, and Staffordshire 50.

No case of smallpox was notified during the week. In the preceding ten weeks 141 cases of smallpox were notified from the outbreak which was centred in Lancashire. The maximum number of cases was 62 in the week ending March 8. Of these cases, 59 were notified in Rochdale C.B.

The number of notifications of acute poliomyelitis was 7 more for paralytic and 1 fewer for non-paralytic cases than in the preceding week. The largest returns were Yorkshire West Riding 4 (Leeds C.B. 2), Lancashire 3, Sussex 3, Nottinghamshire (Southwell R.D.) 2, Glamorganshire (Cardiff C.B.) 2.

A fresh outbreak of dysentery involving 26 persons was reported from Sussex (Hove M.B.). In Norwich C.B. 10 further cases were notified, bringing the total notifications in this city up to 1,321 since the disease first appeared last November. In Lancashire 82 cases were notified, a decrease of 26, and 35 cases in London.

Week Ending May 3

The notifications of infectious diseases in England and Wales during the week included: scarlet fever 877, whooping-cough 3,103, diphtheria 21, measles 4,743, acute pneumonia 582, acute poliomyelitis 24, dysentery 241, paratyphoid fever 10, and typhoid fever 5.

Medical News

Benefits for Pneumoconiosis Sufferers.—The Ministry of National Insurance is now issuing to general practitioners in industrial areas a booklet explaining what to do to help patients suffering from pneumoconiosis (including silicosis and asbestosis) to claim the benefits to which they are entitled. The functions and addresses of the pneumoconiosis medical panels are given, the procedure for arranging *x*-ray examinations is indicated, and the different kinds of benefit are explained. It is emphasized that when a man dies supposedly from pneumoconiosis the coroner should always be informed before completing the death certificate, since a necropsy will probably be necessary before the dead man's dependants will be granted a pension. Further booklets can be obtained from the Ministry of National Insurance, 30, Euston Square, London, N.W.1, or (for doctors in Scotland) from the M.N.I. Central Office for Scotland, 39, Drumsheugh Gardens, Edinburgh, 3.

Terramycin.—The Ministry of Health announces that it has now been decided to make "terramycin" available to medical practitioners in the National Health Service for the treatment of infections with staphylococci resistant to penicillin, in addition to the other illnesses already listed (May 10, p. 1023).

Sanitary Inspectors' Work.—The sanitary inspectors' working party set up by the Minister of Health last year invites anyone interested in its investigations and who wish to submit evidence to write to its secretary, Mr. W. A. Fuller, Ministry of Health, Savile Row, W.1. Any evidence offered should be limited to matters within the working party's terms of reference—the nature of the work at present being done by sanitary inspectors, the nature and functioning of the present arrangements for their recruitment, training, and qualification, and the adequacy of such arrangements.

Medical Association for the Prevention of War.—The first national conference to be organized by the association since its inception last year was held at the Royal College of Surgeons on May 10 and 11. The work was divided into three sessions, preceded by an opening address by Professor F. G. Gregory, F.R.S., Chairman of the National Committee of Science for Peace, who dealt with "The Misuse of Science," and said that secrecy necessarily involved the negation of intellectual freedom. The first session was devoted to "Biological Warfare." Dr. J. H. Humphrey gave an account of the factual historical background, and Dr. M. R. Pollock discussed the ethical problems involved in the misuse of medical science for anti-human and destructive purposes. For the first time in history, biological warfare on a large scale was a thoroughly practical possibility. Silence on the part of the medical profession would be interpreted as tacit approval of the prostitution of medical science. He stressed the necessity for international agreement (involving ratification of the Geneva Protocol by Japan and U.S.A.), the need for the mobilization of informed public opinion, and the necessity that no medical men or women should take part in biological warfare research, in which, incidentally, the claims of defence and offence are inseparable. The second session, on "Psychology of War Preparation," was opened by Dr. J. C. Flugel and Professor L. S. Penrose, who reminded doctors that they could use their prestige to spread knowledge of the mechanisms whereby acts normally rejected as immoral became moral in war. The morning of May 11 was devoted to a discussion on "International Medical Services," opened by Dr. Elizabeth Topley and members of a discussion group of the Birmingham branch of the association.

Aero Medical Association.—The 23rd annual meeting was held on March 17–19 in Washington, D.C. Although primarily an American society it is increasingly international in character. Among the eight new Fellows elected this year for outstanding contributions to aviation medicine were Dr. Kenneth G. Bergin (Bristol), of B.O.A.C., and Dr. G. Edward Hall (London, Ontario), who is president of the University of Western Toronto. Of the three annual awards, the Longacre for outstanding contributions to the psychological or psychiatric aspects of flying went to Sir Frederic Bartlett (Cambridge, England), and the Lyster for general aviation medicine to Dr. Kenneth A. Evelyn (Montreal, Canada). The scientific sessions included a symposium on Space Medicine, in which one paper described the methods and results of experiments which sent mice and anaesthetized monkeys 80 miles up in the stratosphere by rocket.

Applied Statistics.—Since modern statistical methods were largely developed for attacking biological problems, the citadel of medicine soon fell before the onslaught. But many investigators have neither the time nor the aptitude to master the mathematical intricacies of the theory, while yet wishing to apply the methods in practice. A new journal issued by

the Royal Statistical Society (publishers, Oliver and Boyd, Ltd., 98, Great Russell Street, London, W.C.) entitled *Applied Statistics* is intended to meet their needs. Articles in the first number are mainly on industrial and social themes, but some on medical subjects are expected later. The journal also includes a section where questions are answered, and the Editor (Mr. L. H. C. Tippett) invites correspondence. Three issues a year will be published for 25s. (single number 10s.).

Abstracts and Bibliographies.—*Index Bibliographicus* is a guide to those journals which consist entirely or in part of abstracts and bibliographies of relevant literature. Vol. 1 of the third edition, recently published, includes science and medicine, and the medical section lists about 450 journals. It has been prepared by Mr. Theodore Besterman and is published jointly by Unesco and the International Federation for Documentation, and sold by H.M. Stationery Office, price 6s. 6d.

Society for General Microbiology.—At the annual general meeting at Oxford on April 15 the following were appointed for 1952–3: president, Mr. H. J. Bunker; hon. secretaries, Dr. J. G. Davis (general), Dr. E. F. Gale (meetings); hon. editors, Professor B. C. J. G. Knight and Mr. A. F. B. Standfast. In addition to a paper-reading session, a very successful symposium on "The Nature of Virus Multiplication" was held on April 16 and 17 and attended by over 300 microbiologists. This symposium will be published in book form under the editorship of Sir Paul Fildes, F.R.S., and Dr. W. E. van Heyningen. Information about the society may be obtained from Dr. J. G. Davis, 52, London Road, Reading, Berkshire.

Dr. J. H. S. Gear, deputy-director of the South African Institute for Medical Research and assistant director-general of W.H.O., has been awarded the James D. Bruce Memorial Lectureship and Medal by the American College of Physicians.

Emergency Bed Service: Applications and Admissions.—During the seven days ending May 12 the number of applications made by doctors to the London Emergency Bed Service for admission of patients was 930, of whom 90.39% were admitted.

COMING EVENTS

Mind and Body.—The Royal College of Nursing (S.E. Metropolitan Branch) is holding a meeting in the Cowdray Hall, Cavendish Square, W.1, on May 21, at 7.30 p.m. Dr. Noel Harris and the Rev. Jim Wilson will speak on the place of the spiritual factor in medicine.

Guild of St. Luke.—On Ascension Thursday, May 22, at 12 o'clock in Southwark pro-Cathedral, the Right Reverend Cyril Cowderoy, Bishop of Southwark, will celebrate Pontifical Mass for the doctors and their families and nurses of the diocese. Academic robes or uniform may be worn.

West Midlands Physicians Association.—The fourth meeting of the West Midlands Physicians Association will be held at the Royal Hospital, Wolverhampton, on Saturday, May 24, at 11 a.m. There will be general business and case presentations during the morning and scientific communications in the afternoon.

Choir Concert for Cancer Campaign.—The United Hospitals Festival Choir is giving a concert in aid of the British Empire Cancer Campaign at the Albert Hall on May 29, at 7.30 p.m. Brahms's *Requiem* will be sung, and the programme includes Beethoven's violin concerto in D. Tickets from agencies or Mr. G. J. Piller, 92, Shakespeare Road, London, W.7.

Amsterdam Photo Fair.—This exhibition, which is open from June 27 to July 6, is holding a medical-biological day on July 5, an optical day on July 2, and a chemigraphical day on June 28. Medical visitors from Britain will be welcome, either as official representatives or as private persons, and official invitations to the opening, and other information, can be obtained from Professor H. M. Dekking, Oogheelkundige Kliniek, Academisch Ziekenhuis, Groningen, Holland.

106 General Hospital Reunion.—A dinner will be held at 7 p.m. on May 24, at 261, South Lambeth Road, S.W.8. Inquiries to Secretary, 23, Keyes Road, Cricklewood, N.W.2.

SOCIETIES AND LECTURES

A fee is charged or a ticket is required for attending lectures marked ●. Application should be made first to the institution concerned.

Monday, May 19

GUY'S HOSPITAL, London Bridge, S.E.—5 p.m., "*Cardiovascular Surgery*," by Dr. A. Blalock (Baltimore).

●POSTGRADUATE MEDICAL SCHOOL OF LONDON, Ducane Road, W.—4 p.m., "*Gout*," by Dr. George Fearnley.

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—5 p.m., "*Convergence, Convergence Abnormalities, and Heterophoria*," by Dr. T. H. Whittington.

Tuesday, May 20

ROYAL EYE HOSPITAL, St. George's Circus, Southwark.—5 p.m., "*Surgery of Glaucoma*," by Professor Arnold Sorsby.

ST. ANDREWS UNIVERSITY.—At Postgraduate School, Dundee, 5 p.m., "*Problems Connected with Old Age*," by Dr. A. G. Anderson.

ST. GEORGE'S HOSPITAL MEDICAL SCHOOL, Hyde Park Corner, London, S.W.—5.30 p.m., "*Bacteriophage Typing and Epidemiological Problems*," Jenner Memorial Lecture by Brigadier J. S. K. Boyd, F.R.S.

WRIGHT-FLEMING INSTITUTE OF MICROBIOLOGY, St. Mary's Hospital Medical School, London, W.—5 p.m., "*The Pathogenesis of Experimental Pulmonary Anthrax*," by D. W. Henderson, D.Sc., Ph.D.

Wednesday, May 21

GLASGOW UNIVERSITY: DEPARTMENT OF OPHTHALMOLOGY.—8 p.m., "*Experimental Retinal Grafting*," by Dr. Paul Bacsich.

INSTITUTE OF CHILD HEALTH, Hospital for Sick Children, Great Ormond Street, London, W.C.—5 p.m., "*The Treatment of Congenital Heart Disease*," by Dr. A. Blalock (Baltimore).

INSTITUTE OF LARYNGOLOGY AND OTOLOGY, 330, Gray's Inn Road, London, W.C.—5 p.m., "*The Use of A.C.T.H. and Cortisone in Clinical Otolaryngology*," by Dr. S. J. Crowe (Baltimore).

ROYAL COLLEGE OF SURGEONS OF EDINBURGH, Surgeons Hall, 18, Nicolson Street.—3.30 p.m., "*The Development and Growth of the Intervertebral Disk*," 9th Sir John Struthers Lecture by Professor Robert Walmsley.

ROYAL MICROSCOPICAL SOCIETY, Tavistock House (South), Tavistock Square, London, W.C., 5.30 p.m., "*The Electron Microscopic Structure of the Heart Muscle*," by Dr. Bruno Kisch (New York).

Thursday, May 22

BIRMINGHAM UNIVERSITY: FACULTY OF MEDICINE.—At Large Anatomy Theatre, Medical School, 4 p.m., "*Thrombosis and Embolism*," Guest Lecture by Dr. Helen P. Wright.

EDINBURGH UNIVERSITY, University New Buildings, Teviot Place.—(1) 5 p.m., "*Isotopes as an Aid to Diagnosis*," Honyman Gillespie Lecture by Dr. S. Rowlands, B.Sc., Ph.D. (2) At Physiology Class Room, 5 p.m., "*History and Pre-history in Medicine*," lecture-demonstration by Dr. Douglas Guthrie.

EPSOM AND SUTTON INTER-HOSPITAL PSYCHIATRIC ASSOCIATION.—At Mental Hospitals' Group Laboratory, West Park Hospital, Epsom, 5.30 p.m., clinical meeting. Papers and demonstrations.

LONDON UNIVERSITY.—At London School of Hygiene and Tropical Medicine, Keppel Street, W.C., 5.30 p.m., "*Transformation Reactions in Pneumococci*," by Dr. C. M. Macleod (New York). (See also May 29 and June 3.)

●POSTGRADUATE MEDICAL SCHOOL OF LONDON, Ducane Road, W.—4 p.m., "*Simple Lesions at the Cardia*," by Mr. P. R. Allison.

ROYAL EYE HOSPITAL, St. George's Circus, Southwark.—5.30 p.m., "*Cortisone in Ophthalmology*," by Miss M. Savory.

ST. ANDREWS UNIVERSITY.—At Materia Medica Department, Medical School, Small's Wynd, Dundee, 5 p.m., "*Recent Work on Cortisone and Hydrocortisone*," by Professor W. J. E. Jessop.

WEST END HOSPITAL FOR NERVOUS DISEASES.—At 48, Cosway Street, Marylebone Road, London, N.W., 5.30 p.m., "*The Relationship of Psychiatry to General Medicine*," by Dr. Noel Harris.

●WEST LONDON MEDICO-CHIRURGICAL SOCIETY.—At Royal Society of Medicine, 1, Wimpole Street, W., 8.30 p.m., "*The Brain as an Integrated Machine*," Cavendish Lecture by Sir Geoffrey Jefferson, F.R.S.

Friday, May 23

●INSTITUTE OF OBSTETRICS AND GYNAECOLOGY.—At Chelsea Hospital for Women, Dovehouse Street, Chelsea, S.W., 2 p.m., *"The Early Diagnosis of Gynaecological Cancer,"* by Professor G. I. Strachan; 4.30 p.m., *"Pathological Anatomy of the Lymphatic Spread of Cervical Carcinoma,"* by Mr. Stanley Way.

●POSTGRADUATE MEDICAL SCHOOL OF LONDON, Ducane Road, W.—11.15 a.m., surgical clinical-pathological conference; 2 p.m., *"Surgery of the Lymphatics,"* by Mr. J. B. Kinmonth; 4 p.m., *"Malignant Lesions at the Cardia,"* by Mr. P. R. Allison.

Saturday, May 24

●INSTITUTE OF OBSTETRICS AND GYNAECOLOGY.—At Queen Charlotte's Maternity Hospital, Goldhawk Road, Hammersmith, W., 12 noon, *"Exploratory Culdotomy for Tubo-ovarian Physiology at Ovulation Time,"* by Dr. J. B. Doyle (Boston).

APPOINTMENTS

HAY, R. Kenneth, M.D., D.P.H., Medical Officer of Health and Divisional Medical Officer, Urban and Rural Districts of Nantwich, Cheshire County Council.

HOSPITAL FOR SICK CHILDREN, Great Ormond Street, London, W.C.—*Part-time Registrar to Plastic Department*, T. J. S. Patterson, F.R.C.S. *House-physicians*, K. Hugh-Jones, M.D., M.R.C.P., and A. P. Roberts, M.R.C.P., D.C.H. *House-surgeon*, M. B. N. J. O'Donnell, M.B., B.Ch.

LEWIS, W. J., M.R.C.S., L.R.C.P., D.A., Part-time Consultant Anaesthetist, Mid-Glamorgan Hospital Management Committee Area, Welsh Regional Hospital Board.

LIVERPOOL REGIONAL HOSPITAL BOARD.—*Consultant Paediatric Surgeon to Alder Hey Children's Hospital*, P. P. Rickham, M.S., F.R.C.S., D.C.H. *Part-time Anaesthetist to Hospitals in Liverpool*, John Moore, M.B., B.Ch., D.A. *Part-time Consultant Anaesthetist to Hospitals in Central Wirral and Chester Groups*, Gough Hughes, M.B., Ch.B., D.A. *Part-time Consultant Anaesthetist to Hospitals in Southport and Ormskirk Groups*, C. P. Brown, M.B., Ch.B., D.A. *Whole-time Consultant General Surgeon to Hospitals in North Liverpool Group*, A. F. Murphy, Ch.M., F.R.C.S. *Whole-time Consultant Physician-Superintendent to Walton Hospital*, A. Skene, M.B., Ch.B., M.R.C.P.Ed. *Whole-time Assistant Psychiatrist to Walton Hospital*, E. T. Downham, M.B., Ch.B., D.P.M. *Whole-time Senior Resident Medical Officer to Barrowmore Hospital*, J. T. Foody, M.B., B.Ch.

MCCALL, JAMES JACK, *M.C.*, M.B., Ch.B., D.P.H., Senior Resident Medical Officer, Erskine Hospital, Bishopton, Renfrewshire.

MCDOUGALL, JOHN, M.B., Ch.B., F.R.C.P.Ed., Dip.Psych.Ed., Physician-Superintendent, Perthshire Mental Hospitals (Murray Royal and Murthly Hospitals).

MACQUEEN, IAN A. G., M.D., D.P.H., Medical Officer of Health, City of Aberdeen.

SOUTH-WEST METROPOLITAN REGIONAL HOSPITAL BOARD.—*Whole-time Deputy Physician-Superintendent and Consultant Psychiatrist, Tooting Bec Hospital*, M. A. Walsh, L.R.C.P.&S.I., D.P.M. *Whole-time Deputy Physician-Superintendent and Consultant Psychiatrist, Park Prewett Hospital*, L. Ford, M.D., D.P.M. *Whole-time Physician-Superintendent and Consultant Psychiatrist, St. James's Hospital, Portsmouth*, W. L. Milligan, M.D. *Part-time Consultant Anaesthetist, Western Area Thoracic Surgical Unit*, E. S. Machell, M.B., Ch.B., D.A. *Part-time Consultant Ear, Nose, and Throat Surgeon, Farnham Group of Hospitals*, D. Bartlett, F.R.C.S. *Part-time Consultant Obstetrician and Gynaecologist, Farnham Group of Hospitals*, S. S. F. Pooley, M.B., B.S., M.R.C.O.G. *Pathological Registrar, Portsmouth and Isle of Wight Groups Service*, J. MacI. Moore, M.B., Ch.B. *Pathological Registrar, Royal Hants County Hospital*, R. F. Pearson, M.B., B.Chir. *Pathological Registrar, South London Hospital*, Rhiannon Shorter, M.R.C.S., L.R.C.P. *Infectious Diseases Registrar, Western Hospital*, E. P. O'Sullivan, M.D. *Psychiatric Registrar, Park Prewett Hospital*, P. H. Rogers, M.B., B.Chir., M.R.C.P. *Senior Surgical Registrar, St. James' Hospital, Balham*, T. G. E. Loosemore, M.Chir., F.R.C.S. *Surgical Registrar, Putney Hospital*, A. G. Parks, M.D., F.R.C.S., M.R.C.P. *Medical Registrar, Royal Surrey County Hospital*, H. R. C. Riches, M.B., B.S. *Medical Registrar, Croydon General Hospital*, K. G. Neill, M.B., Ch.B. *Orthopaedic Registrar, Salisbury General Hospital*, D. J. Ghadialy, M.B., B.S. *Senior Medical Registrar, Kingston Hospital*, R. S. Crow, M.R.C.P. *Medical Registrar, St. Mary Abbots Hospital*, Elizabeth Williamson, M.D.

BIRTHS, MARRIAGES, AND DEATHS

DEATHS

Blatchford.—On April 27, 1952, in a nursing-home, Bournemouth, William Nichols Blatchford, M.R.C.S., L.R.C.P., Surgeon Commander, R.N., retired.

Collen.—On April 27, 1952, at The Limes Nursing Home, Ditchling Common, Burgess Hill, Sussex, Edward Victor Collen, M.D., aged 76.

Grattan.—On April 30, 1952, at the Lister Hospital, Hitchin, Herts, Henry William Grattan, C.B.E., D.S.O., M.R.C.S., L.R.C.P., D.P.H., Colonel, late R.A.M.C., retired, late of 6, Sollershott West, Letchworth, Herts, aged 80.

Kingston.—On April 29, 1952, William George Kingston, L.M.S.S.A., Major, I.M.S., retired, of Great Bedwyn, near Marlborough, Wilts.

Pearson.—On May 2, 1952, at the Southern Hospital, Dartford, Kent, Malcolm George Pearson, B.M., B.Ch., D.T.M.&H., aged 49.

Robertson.—On April 29, 1952, at Ryecroft Burton, Carnforth, Westmorland, William Howell Robertson, M.B., C.M., aged 82.

Sharrard.—On April 25, 1952, in London, Ethel Margaret Sharrard, M.B., Ch.B., of 22, Hathaway Gardens, London, W., aged 35.

Steven.—On April 29, 1952, at 8, Park Street, Ripon, Yorks, Peter Alexander Steven, M.B., Ch.B., aged 74.

Tordoff.—On April 28, 1952, at 35, Dallam Avenue, Morecambe, Lancs, Bertram Tordoff, M.B., B.Ch., formerly of Bradford, Yorks.

Towers.—On April 29, 1952, Arthur Henry Towers, M.B., Ch.B., B.Hy., D.P.H., of Brampton, Cumberland, aged 65.

Wales.—On May 3, 1952, at his home, Forest Lodge, Southwold, Suffolk, Edward Garneys Wales, M.B., B.Ch., late of Downham Market, Norfolk, aged 75.

Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Sodium-containing Barbiturates in Eclampsia

Q.—*Is there any objection to the use of soluble barbiturate compounds—i.e., those containing a sodium atom in their molecule—in the treatment of pre-eclampsia when one is also reducing the intake of sodium, as sodium chloride, in the diet?*

A.—The disadvantage of using soluble barbiturate compounds containing sodium can be overcome by giving soluble phenobarbitone, which is free from sodium. The indication for administration by the parenteral route would be in cases of fulminating pre-eclampsia or eclampsia and would be for a short time. In these cases rectal bromethol or paraldehyde has the advantage of rapidity of effect. These solutions do not contain sodium. In less severe cases of pregnancy toxaemia phenobarbitone or amylobarbitone can be given by mouth. There is no sodium in these preparations.

Constipated Infant

Q.—*My son, aged 2 years 4 months, has a motion once every four or five days in spite of the trial of various aperients. Naturally the huge motion is very painful when it is passed, and I am very worried about it, although I try not to let him realize this. In other ways he appears quite healthy; he is tall for his age and weighs 31 lb. (14 kg.). He has a very small appetite. Can you please advise what should be done?*

A.—It is not uncommon for children at this age to have infrequent motions. Particularly when the appetite is small and the child healthy, most of the food ingested is absorbed and there is insufficient residue to provide enough bulk for a regular daily motion.

Treatment should be directed to increasing the intake, both fluid and solid, and to providing adequate roughage and a proportion of easily fermentable carbohydrate. Appetite may be increased by yeast extracts, either in sandwiches or on buttered biscuit. Stewed apples or mashed prunes at or before breakfast make a good start to the day. Extract of malt in doses of 1 to 2 dr. (3.5 to 7 ml.) two or three times a day is as good a laxative as any. A cupful of sweetened orange juice may be given each afternoon, and honey sandwiches at tea-time may also be helpful.

Treatment of Lightning Pains

Q.—*How may typical tabetic lightning pains in the legs be relieved? They have not responded to two intensive courses of penicillin and mist. pot. iod., and about 12 injections of bismuth.*

A.—Many people have for a great many years been seeking the answer to this question. Presumably the usual analgesics have been tried without success; it may be necessary to have recourse to morphine, but the danger of addiction must be kept in mind. It might be worth while trying the effect of fever treatment induced by intravenous T.A.B. vaccine injections. B. T. Horton (*Postgrad. Med.*, 1951, **9**, 1 and 88) reports relief of agonizing pain, which had been present for 32 years, by histamine injections. In the last resort, and if the pains are severe and intractable, making the patient's life a misery, prefrontal leucotomy might be considered.

Absence of Sensation during Orgasm

Q.—*I have a patient—a married man aged 26—who was married six months ago. Since his marriage he has been able to have normal intercourse with his wife, usually about six times weekly. He satisfies his wife in all respects, but does not get an orgasm himself. He has a normal ejaculation but no accompanying pleasurable sensations. He is naturally very worried about this state of affairs, and his standard of work has deteriorated. Could you advise a solution to this problem, please?*

A.—All that can be said with any confidence in answer to this question is that the lack of sensation is unlikely to be explained by anything physically wrong with this patient. Physically speaking, he is likely to be a normal man and sexually developed. The emotional life and the appreciation of sensations are closely linked, and his lack of satisfaction probably has a psychological explanation. There are, for example, men who remain at the auto-erotic stage of sexual development and who obtain far more satisfaction and sensation from masturbation than from adult sexual intercourse. This case must be investigated from the psychological standpoint before a precise diagnosis can be made.

Sensitivity to Oak

Q.—*May I please have your advice on the following problem? I have several patients who give practically identical histories of what I presume is a sensitivity to oak trees. They all say that the symptoms arise only if they handle, cut, or saw green oak—i.e., standing wood—and that the symptoms are worse if the tree is in leaf. Briefly, the symptoms consist of a burning irritation of exposed parts (hands, wrists, and face) with or without a fine weeping eczema-like condition on parts of the face and neck and sometimes on the hands; one case produces an urticaria-like swelling of eyebrows and lids as well. They all say they realize that they must keep away from this wood, and yet in a country labourer this is not feasible. Antihistamine drugs seem to have little effect on the condition, which lasts from three to seven days as a rule.*

A.—Sensitivity to green oak is a well-recognized contact allergy. Prophylactic measures are the most important. These consist in protection of the hands and arms from the sap by the use of a water-repellent barrier cream applied before work and renewed after four hours. At the same time the men must be taught not to mop their faces with handkerchiefs which they have contaminated by touching, and not to touch their faces with their hands. The dermatitis is purely a contact one. Calamine lotion or liniment usually gives most relief when the dermatitis has occurred.

Periodic Vulval Oedema

Q.—*A girl of 14 at each period suffers from severe oedema of the labia minora. The oedema starts on the fourth day of a heavy seven-day period, and is sufficient to prevent walking for 24 hours. Antihistamine drugs have no effect, and testosterone resulted in epimenorrhoea. Can you suggest a likely diagnosis and treatment?*

A.—From the late time of onset of the oedema and the fact that the loss is heavy, it would seem most likely that the cause is chafing by soiled sanitary towels. Other contributory factors may be infrequent changing of the towels and a failure to bath during menstruation, while if the labia minora are long and protruding they are more likely to suffer friction and become oedematous. One possible solution is to employ intravaginal tampons instead of vulval pads, but the patient is rather young to adopt this method. The oedema would therefore best be avoided by scrupulous attention to local hygiene during menstruation. The vulva should be bathed daily and dried carefully afterwards. Superior quality sanitary towels of the type with a "knitted back" should be used and they should be changed frequently. In addition undue activity and standing for long hours should be avoided during the period. Finally, if the labia minora are excessively long and if all else fails, plastic surgery might be considered as a last resort.

Bleeding from the Umbilicus

Q.—*What are the causes and treatment of intermittent bleeding from the umbilicus in small children?*

A.—In the first month of life umbilical bleeding is usually from a blob of granulation tissue remaining from the cord stump. The umbilicus is best examined with a large electric auriscope and such a granuloma cauterized with a "silver stick." If the granuloma recurs or appears to be big, it is likely that the "raspberry" tumour is intestinal mucosa from a Meckel's remnant. Again, it should be cauterized, or, if it is pedunculated, a piece of thread may be tied round its base. More complete forms of vitello-intestinal remnant need surgical excision.

In older children, infection and foreign bodies lead to excoriation of a deep umbilicus. Fungus infections and scabies are common in the umbilicus. The former, sometimes presenting as bleeding, are best treated by ionization with copper sulphate.

NOTES AND COMMENTS

Post-mortem and Ante-mortem Caesarean Section.—Dr. J. W. MAGNER (Cork) writes: In "Any Questions?" (April 19, p. 879) your expert states that "Caesarean section carried out to recover a live child from a dying woman has been carried out occasionally since at least 1603, but is generally abhorrent to both medical attendant and relatives. It is a proper procedure according to the Roman Catholic faith. . . ." This statement of Roman Catholic teaching is incorrect. Thomas A. Jorio, S.J., Professor of Moral Theology in the Pontifical Faculty of Theology in Naples (*Moral Theology*, 1946), states: "Caesarean section on a living mother can never be licitly carried out, if proximate danger to the life of the mother will arise from the operation, and this is true even in the case where, if the operation be not carried out, both mother and child will certainly die."

We referred our correspondent's letter to Father A. BONNAR, O.F.M. (Cambridge), who writes: The principles involved are: (1) It is never permissible to kill one person in order to save the life of another, nor is it permissible for anyone to kill himself (or herself) or to direct himself (or herself) to be killed for that purpose. (2) It is, however, permissible, and may in some cases be obligatory, to expose one's life to serious danger to save the lives of others or minister to their other needs. Examples of this would be a person going into a burning building to rescue people trapped there; or doctors, nurses, and priests tending the sick even during a highly infectious epidemic. Applying these principles to caesarean section on a dying woman, we get the following conclusions: (1) If her condition is such that the procedure really and truly amounts to killing her, then it is not permissible. (2) If the operation, however, merely means seriously endangering her life, then the operation is permissible. But the mother is not under a moral obligation of allowing it. The reason for this last assertion is that, though the mother must take all reasonable and natural precautions to save her child, she is not obliged to go to the length of deliberately and positively putting her own life in danger by resorting to this out-of-the-normal procedure. I assume throughout that the foetus is viable.

All communications with regard to editorial business should be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1. TELEPHONE: EUSTON 4499. TELEGRAMS: *Aitiology, Westcent, London.* ORIGINAL ARTICLES AND LETTERS forwarded for publication are understood to be offered to the *British Medical Journal* alone unless the contrary be stated.

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